HEY GROVES'

SYNOPSIS OF SURGERY

ILLUSTRATED

EDITED BY

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PREFACE TO THE TWELFTH EDITION

It is no easy matter to follow in the footsteps of a surgeon of the calibre of Hey Groves, and yet the revision of this well-known Synopsis has been a labour of love, for Hey Groves was a friend and a surgical father to me. It was a sad day in November when this revision was almost completed that Groves died after a long illness His departure leaves a great gap in the ranks of British surgeons. The inclusion of his portrait in this edition will be welcomed by all who scan its pages; it depicts Groves in the cheery mood which was such a stimulus and example to all

The whole text of this edition has been very carefully revised and brought up to date. The inclusion of penicillin and the sulphonamide group of drugs in the treatment of wounds and infections has necessitated a re-writing of some chapters.

Modern methods of radium therapy have been incorporated, while some of the old amputations which are no longer in use have been omitted.

In the work of revision I have been ably assisted by my friend and colleague, Surgeon Lieut.-Commander R. A. Mogg, F.R.C.S., to whom and to Miss Pillers, who has done a number of new illustrations, my most grateful thanks are due.

My thanks are also due to the publishers for their unfailing courtesy and constant aid in seeing the book through the press at a very difficult time.

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FROM THE PREFACE TO THE FIRST EDITION

THE title of this small book describes its aim and scope. In recent years the tendency of all surgical literature to grow in bulk and diversity has of necessity compelled authors to enlarge their text-books more and more. Complete treatises on surgery occupy many volumes, and even the most concise require months for their perusal, so that a student often despairs of being able to revise his knowledge.

The present volume is an attempt to make an epitome of the salient facts in surgical practice, and to place these facts in such a manner that they may most easily and rapidly be referred to or revised It has been compiled almost entirely from notes made by the author in preparing students for examinations

It is of course obvious that the large works cannot be dispensed with; but after a student has carefully read through a complete text-book, which ought to be done at the time he is actually engaged in ward and out-patient work, he may find this Synopsis aid his memory in retaining a vast array of facts in an orderly manner.

The busy practitioner, who is expected to keep in practical touch with an ever-advancing science, has too often to allow the big books to go unread, whilst magazine articles are disconnected, discursive, and difficult for reference. It is thought that this synopsis may, by its methodical arrangement, prove of assistance by presenting the diagnosis and treatment of surgical conditions in a concise manner.

The special arrangement of headings, type, and indented margins enables the reader to see the scope of the subject at a glance, and to refer to any part of it at will. It may also offer some suggestions as to the arrangement of answers in a clear and succinct manner at examinations.

Only the main points of operations are referred to, so that the principles which underlie each procedure may not be lost in a mass of details.

Anatomical and pathological facts are only inserted when they are essential to a comprehension of clinical signs and operative measures, but a summary of surface markings is added in the last chapter, which includes all those of importance.

E. W. H. G.

CLIFTON, *July*, 1908

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SYNOPSIS OF SURGERY

CHAPTER I

INFLAMMATION AND SUPPURATION

INFLAMMATION

Definition.—The succession of changes which occur in living tissue when it is injured, provided the injury is not of such a degree as at once to destroy its structure and vitality.

Causes.—(1) Mechanical injuries; (2) Bacterial invasion; (3) Thermal injuries—heat or cold; (4) Chemical injuries; (5) Electrical injuries—high-tension currents—X rays.

Histological Changes.—

1. VASCULAR CHANGES —
   - HYPERÆMIA.—The small vessels dilate—The blood-stream slows after momentary acceleration.
   - STASIS.—The blood-stream comes to a standstill—The blood clots in the vessels.
   - EXUDATION.—The white cells, which have been hugging the vascular wall, now creep through it—The red cells escape passively from the vessel—The serum of the blood oozes out from the vessel.
2. TISSUE CHANGES.—Vary with the nature and virulence of the infective agent, with the resistance of the individual, and with the local circulatory conditions.

   May be one of three processes, viz.:—
   - *a*. PROLIFERATION.—Results from mild or chronic infections. The connective and endothelial cells multiply by division. Fresh connective tissue is formed thereby. Produces thickening, fibrosis, or sclerosis.
   - *b*. GRANULATION.—Produced by trauma, e.g., open wounds, mild infection, or when virulent infection is at an end Exuded lymphocytes form a mass of 'small round cells', which invades and replaces the tissues.
   - *c*. DESTRUCTION.—
     - i. Colliquative necrosis. The result of non-bacterial injury, especially burns.

       Excessive fluid exudation, with separation and disintegration of cells, forms a blister.
     - ii. Coagulation necrosis. The result of infection by pyogenic organisms.

       The exuded serum containing bacteria and toxins coagulates round the tissue cells. Many of the tissue cells and leucocytes are killed by the toxins.

Inflammation—Tissue Changes—Destruction, ***continued.***

Ends in one of four ways:—

1. Catarrh.—The dead cells are thrown off from a free mucous surface, but only the superficial parts are lost.
2. Ulceration.—The dead cells are thrown off from a free surface in such numbers as to leave an area devoid of skin or mucous membrane.
3. Suppuration.—The coagulated tissue is peptonized and liquefied by the bacteria and leucocytes. Polynuclear leucocytes destroy and are destroyed by the bacteria.
4. Sloughing or gangrene.—The tissue dies *en masse* and forms a dead slough.

Local Results.—

1. RESOLUTION without any permanent effect, only possible when the process has stopped short of thrombosis.
   The circulation is resumed.
   The exuded cells and serum are absorbed.
2. ORGANIZATION of the inflammatory products
   Only occurs when the infective agent is mild.
   The granulation tissue or 'small round cells' and the connective-tissue cells form fibroblasts, and these form new connective tissue.
   New vessels are formed by a budding out from old vessels.
3. DESTRUCTION of the tissues, usually followed by repair by granulation tissue.
   ULCER.—The loss of tissue on a free surface.
   ABSCESS.—The liquefied dead tissues below the surface.
   SLOUGH.—A piece of tissue which has been killed *en masse*, but not liquefied.
   GANGRENE.—The death of tissues in massive quantity A term usually applied to the death of an organ or part of a limb.
   N.B.—Ulceration, sloughing, or gangrene may be caused by other things besides inflammatory infections.

Signs and Symptoms.—

GENERAL.—

Inflammatory fever usually present.
Temperature raised, pulse-rate correspondingly increased.
All organic functions tend to be impaired.
Muscular and nervous sustems—malaise, mental and muscular inadequacy, or prostration.
Secretory functions diminished—anorexia, indigestion, constipation.
Excretory functions—lowered urinary output The increased metabolism increases output of urea and urates.
All the above vary according to the nature of the infection and the resistance of the individual.
In the absence of infection—for example, in traumatic cases—any effect is due to substances produced from dead or damaged tissues.

LOCAL.—

1. *Heat*—caused by hyperæmia; valuable clinical sign indicating active changes proceeding.
2. *Redness*—caused by hyperæmia. If part affected is deep, may be invisible; even then surface veins may be unduly prominent.

3. *Swelling*—in part the result of exudation, and in part due to hyperæmia. The maximum swelling may not coincide with the site of inflammation.
4. *Pain*—due to increased tension in the part affected; elevating the part may lessen tension and pain. Pain is increased by pressure and movement. Sometimes pain may be referred to a site distant from the inflammatory process.
5. *Loss of function*.

Treatment of Acute Inflammation.—

LOCAL.—

Remove the exciting cause if possible. Rest the inflamed part.

Rest should be thorough and continuous.

ENCOURAGEMENT OF HYPERÆMIA to hasten natural process. Hot fomentations. Radiant heat, infra-red and ultra-violet light. Short-wave therapy.

PASSIVE HYPERÆMIA in acute inflammation is obtained in three ways: (1) By an elastic bandage tied round a part at a distance from the inflamed focus. This should be tight enough to obstruct the venous return without affecting the arterial pulse, and should relieve, not increase, the pain. It is left on for twenty hours out of twenty-four at first, and for shorter periods later. (2) By cupping-glasses, which are exhausted by an attached rubber ball or suction pump. These are placed over the affected focus when situated on the trunk. They are applied for about one hour a day, the suction being released every five minutes for one minute.

CONSTITUTIONAL.—

Rest in bed. Low diet.

Antitoxic sera to neutralize the toxins.

Antibacterial sera to kill the bacteria.

Inoculation with modified toxins to increase opsonins.

Chemotherapy.

Treatment of Chronic Inflammation.—

Remove the cause if possible. Rest

ACTIVE HYPERÆMIA: Massage—Hot-air treatment.

COUNTER-IRRITATION: Blister—Actual cautery—Iodine or mercurial applications.

PRESSURE: Bandaging—Elastic pressure—Scott's dressing—Unna's paste.

INJECTION TREATMENT.—Alcohol or saline, e.g., in affections of sensory nerves.

SUPPURATION

Definition.—The molecular necrosis and liquefaction of living tissues due to pyogenic bacteria

Varieties.—

RATE OF DEVELOPMENT.—Acute or chronic.

NATURE OF INFECTION.—Simple pyogenic infection.

Specific infection with the bacteria of a specific fever, e.g., typhoid, tubercle.

SITUATION.—

MUCOUS MEMBRANE: Catarrh, e.g., acute otitis media.

SEROUS MEMBRANE: Purulent serositis, e.g., peritonitis or empyema.

Suppuration—Varieties—Situation, ***continued.***

FREE SURFACES: Ulceration—'Inflamed ulcers.'

BELOW SURFACE: Abscess—localized suppuration; Cellulitis—diffuse suppuration.

Cause.—Infection by bacteria:—

Through a wound.

Through the lymph or blood-stream.

Bacteria which cause suppuration (*see Fig.* 1).—

I. SIMPLE PYOGENIC BACTERIA, i.e., those which produce suppuration without causing any specific fever.

1. STAPHYLOCOCCUS PYOGENES.: *aureus*, *albus*, and *citreus*.
   - Grow readily in culture media.
   - Form luxuriant patches in a few days.
   - Gram-positive.
   - Liquefy gelatin.
   - Powerful tryptic action.
   - Form clusters of minute dots in the growth or tissues.
   - Are the commonest cause of suppuration.
   - The *aureus* is the most virulent. The *albus* least virulent.
   - No antitoxin.
   - Useful vaccine.
   - Common cause of:—
     - Boils, superficial abscesses; Carbuncles (here a slough is produced —*see* p. 135)—Periostitis—Perinephric abscess—Empyema—Osteomyelitis and arthritis
2. STREPTOCOCCUS PYOGENES.—
   - Forms chains of minute dots.
   - Grows slowly outside the body
   - Forms minute dot-like colonies.
   - Gram-positive.
   - Survives desiccation.
   - Hæmolytic and non-hæmolytic. (Former more virulent.)
   - Does not liquefy gelatin.
   - Antiserum is used.
   - Causes virulent suppuration: Erysipelas—Cellulitis—Acute arthritis —Peritonitis—Empyema—Septicæmia and pyæmia.
3. BACILLUS COLI COMMUNIS.—
   - Occurs in the healthy intestine.
   - Is a motile bacillus with flagella.
   - Grows readily in culture media.
   - Gram-negative.
   - Produces Indol—Free gas—Acid.
   - Gives rise to specific agglutinins.
   - Causes acute peritonitis—Cystitis—Pyelitis—Any abdominal suppuration—Cholecystitis—Rarely abscesses elsewhere in bones, joints, or soft parts.
4. BACILLUS PYOCYANEUS.—
   - Motile with flagellum.
   - Grows in wounds—Produces greeny-blue pus.
   - Gram-negative.
   - When it occurs in a superficial wound its toxic action is only slight.
   - When very virulent, it may cause peritonitis, septicæmia, or pyæmia.
   - Especially found in long-standing sinuses of bone.

II. SPECIFIC PYOGENIC BACTERIA.—**Bacteria which produce a well-marked specific disease, and occasionally local suppuration.**

1. PNEUMOCOCCUS.—
   Stains by Gram's method.
   Is a diplococcus. Usually encapsuled.
   Grows outside the body with difficulty
   Is the ordinary cause of pneumonia.
   Also causes: Empyema: common. Peritonitis, Arthritis, Osteomyelitis: rare.
2. BACILLUS TYPHOSUS.—
   Does not stain by Gram's method.
   Forms specific agglutinins.
   Does not produce indol or gas.
   A multiflagellate bacillus.
   Is the ordinary cause of enteric fever.
   May cause:—Bone suppurations—periostitis. Suppuration in the gall-bladder. Local peritonitis.
3. GONOCOCCUS (*see* p. 40).
4. TUBERCLE.

Minute Anatomy of an Abscess.—

I. IN THE EARLY STAGES it shows all the signs of acute inflammation, ending in coagulation necrosis.

II. IN THE FULLY MATURED ABSCESS the central portion has become liquid pus.

The wall from without inwards consists of four zones.—

1. HYPERÆMIA and exudation.
2. THROMBOSIS with round-celled infiltration.
3. 'SMALL ROUND CELLS' with many bacteria.
4. Central collection of PUS.

III. IN THE HEALING ABSCESS a zone of fibrosis intervenes between the granulation tissue and the healthy tissue.

The innermost layer consists of granulation tissue, i e., small round-celled tissue permeated by new blood-vessels.

Bacteria are practically absent, or dead.

Nature of Pus (*see Fig.* 2).—Fluid of sp. gr. 1030.

Contains 85 per cent water—Opaque white or yellow colour—Alkaline in reaction. The liquid portion consists of a solution of peptones.

The solid portion consists of: Dead polynuclear leucocytes—Living leucocytes—Bacteria—Debris of partially digested tissues.

Signs and Symptoms of Acute Suppuration.—

SIGNS OF ACUTE INFLAMMATION, and especially:—

FLUCTUATION—showing the fluidity of contents.
ŒDEMA of the skin—of great value in deep suppuration.
HARD BRAWNY TEXTURE.
PAIN becoming distinctly THROBBING.

GENERAL SIGNS are those of a bacterial infection, and especially:

RIGOR or shivering fit, with rapid rise of temperature.
LEUCOCYTOSIS. Leucocytes increase from 5000 to anything up to 100,000 per c.mm.
Absent in severe fatal infections.
It indicates local infection well resisted.

Suppuration, *continued.*

Treatment of Acute Suppuration.—

1. IN EARLY STAGES when suppuration is doubtful, treat as for acute inflammation.
2. WHEN PUS IS CLEARLY INDICATED.—
   Open freely from the surface
   Open all secondary loculi into the main cavity.
   Make the opening in the most dependent position to allow free drainage.
   Make as many openings as are necessary to drain the cavity freely.
   Swab out the pus or irrigate with antiseptics.
   Do not scrape the wall, for fear of infecting fresh tissues.
   Put on an antiseptic dressing. Drain with tubes or gauze.
   Continue fomentations or passive hyperæmia.

Chronic Abscess.—

DEFINITION.—A collection of pus without the signs of inflammation. Usually of very slow development

PATHOLOGY —

TUBERCLE is the cause in the vast majority of cases.

PYOGENIC COCCI may give rise to an abscess, and then the organisms die or become latent, and the abscess remains cold and chronic. Especially in connexion with bones—Brodie's abscess.

ANATOMY.—

May be connected with any tuberculous focus

Commonly in connexion with BONE, JOINT, or LYMPHATIC disease.

Ceases to be a chronic abscess when it is infected with active pyogenic organisms. Hence the predilection for sites removed from skin or mucous membrane.

GROWTH: Slowly spreads along lines of areolar tissue Guided by fascial planes, or the sheaths of blood-vessels

WALL: Usually thick and well defined. Lined by a layer of soft granulations which forms 'the pyogenic membrane' These contain tubercle bacilli and giant-cell systems which form caseous masses, in the tuberculous cases A dense layer of fibro-cicatricial tissue lies outside the granulations

CONTENTS: Pus of a special character (*see Fig* 3). Curdy and fatty debris. Very few cells, and these degenerate. Occasionally cholesterin crystals No bacteria except tubercle, and those very scanty. Pus is usually infective to animals

COMMON EXAMPLES of Chronic Tuberculous Abscesses.—

PSOAS ABSCESS —Caused by dorsi-lumbar tuberculous disease.

Spreads beneath the internal arcuate ligament. Guided by the sheath of the psoas muscle. Forms a fluctuating swelling above Poupart's ligament on the outer side of vessels. Dips beneath the femoral vessels, and enters the thigh behind and on the inner side. May continue down the thigh to the knee.

Aspirate over the fluctuating areas. Usually.—
a. Above the outer half of Poupart's ligament, or
b. Below the inner half of Poupart's ligament, or
c. In the loin through the quadratus lumborum.
Occasionally at two of these places.

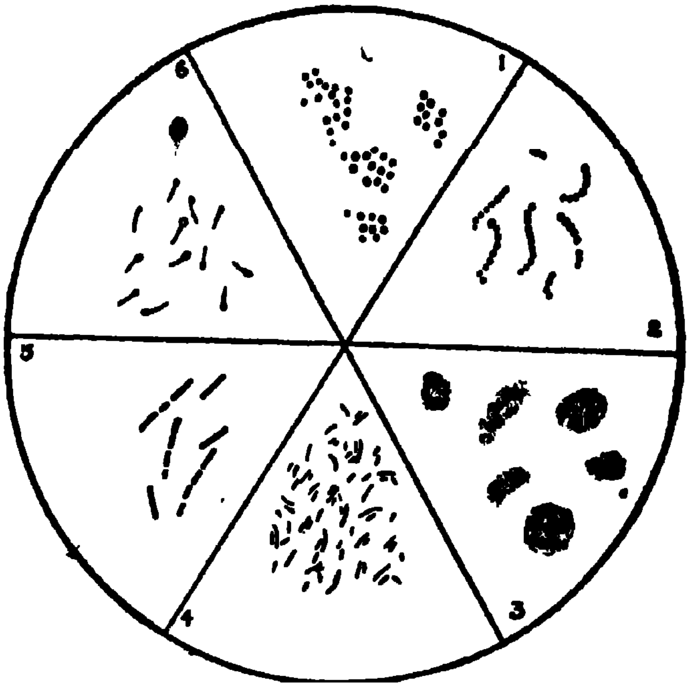

Fig. 1.—Various forms of bacteria.
1. Staphylococci, 2, Streptococci, 3, Gonococci, 4, *B coli*; 5, *B. anthracis*; 6, *B. tetani*.

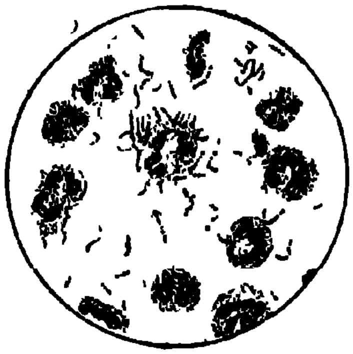

Fig. 2.—Pus from acute abscess, showing cocci and leucocytes.

Fig. 3.—Pus from chronic abscess, showing tubercle bacilli. All cells are disintegrated.

Chronic Abscess—Common Examples, ***continued.***

Iliac Abscess.—Arises from disease of the ilium, sacro-iliac joint, or of the hip-joint through the acetabulum. Forms a fluctuating swelling in the iliac fossa Aspirate or open above outer half of Poupart's ligament.

Lumbar Abscess.—Usually caused by disease of the lumbar vertebræ. Is directed by the dorsal branches of the lumbar arteries. Tracks beneath the psoas and on inner side of quadratus. Points on outer side of the erector spinæ.

Aspirate or open by a vertebral incision over the outer border of erector spinæ.

Costal or Intercostal Abscess.—Caused by disease of the ribs or of the dorsal or cervical vertebræ.

Aspirate or open over the fluctuating swelling.

Chronic Retropharyngeal Abscess—Caused by disease of the upper cervical vertebræ.

Lies behind the prevertebral fascia.

Open behind the sternomastoid.

TERMINATION.—

1. **Recovery** by absorption of the abscess contents
   The contents become caseous, or calcified, or are absorbed
   A fibrous scar remains.
   Seldom happens except in very small abscesses
2. **Bursting** externally or into a mucous canal
   Rapidly infected with pyogenic organisms.
   Incurable or fatal sepsis results.
3. **Death from Septic Absorption** after pyogenic infection.
   Usually of a hectic type.
4. **Death from Amyloid or Lardaceous Disease** after pyogenic infection. Waxy degeneration occurs in the vessels and viscera, viz.: in the arterioles and capillaries; in the liver, spleen, kidneys, and intestines.
   Reactions of the Waxy Substance.—
   Iodine—turns brown Sulphuric acid after iodine—inky blue
   Methyl violet—red.
   Symptoms and Signs.—
   Enlargement of the liver and spleen. Albuminuria, with great increase of amount of urine, with casts. Diarrhœa.
   Condition can be arrested if the radical removal of the suppurating focus is done early.
5. **Recurrence** after spontaneous absorption or after operation is very common.

SYMPTOMS AND SIGNS.—

Swelling of slow appearance.

Absence of heat, redness, tenderness, or raised temperature.

Fluctuation is well marked unless the abscess is very deep.

Other symptoms and signs are merely those of the cause of the abscess, e.g., signs of caries of the spine or of sacro-iliac disease.

Sepsis, acute or chronic, only develops after pyogenic infection, i.e., after the abscess has opened in the majority of cases.

Amyloid disease may also occur.

TREATMENT.—

Excision.—If anatomically possible—e.g., in lymph glands, or rarely in an early rib abscess.

Aspiration.—This should be the routine method in most cases. A large needle is thrust through healthy skin after the latter has been pulled down, so as to make a valvular opening. Evacuate by simple pressure or an aspirator.

Injection of Liquefying Solutions.—If the contents are too thick to run through a needle, a fluid—e.g., thymol 1 part, camphor 2 parts, and ether 3 parts—is injected, and then the aspiration is repeated in a week.

Injection of Antiseptics.—After aspiration an emulsion of iodoform (10 per cent in glycerin or ether) is injected.

Incision and Scraping.—Abscess is opened through a small valvular incision made in healthy skin. If the skin is unhealthy, thin, or red, it should be excised. The pus is evacuated. Wall of abscess is scraped with a sharp spoon. Any loose fragments of bone are removed. Incision is carefully sutured.

This method is especially indicated where there are accessible sequestra.

Evacuation and Packing.—As in the last method, but operation is completed by packing with gauze smeared with bismuth iodoform paraffin paste (B.I.P.P.) after swabbing out with alcohol. Place deep sutures but do not tie. Remove pack in one or two days and tie sutures.

Especially suitable for large abscesses where the whole cavity is accessible.

Rest and Immobilization of the Affected Area.

General Treatment, especially sanatorium treatment, to improve general body health.

CHAPTER II

SINUS, FISTULA, AND ULCERS

Definition of Sinus and Fistula.—A sinus is a track lined by granulations, leading from a free surface into the depths of the tissues. A Fistula is a track which opens at both ends on to an external or internal cutaneous or mucous surface.

Structure.—An outer layer of fibrous tissue, which in old cases becomes very thick and dense like cartilage. An inner layer of granulations, usually infected by bacteria.

Causes.—Abscess—Penetrating wounds.

CAUSES OF NON-CLOSURE of sinuses and fistulæ.

- Dead septic body in the sinus, e.g., necrosed bone.
- Foreign body which is septic, e.g., ligature.
- Insufficient opening of a deep cavity.
- Inability of a deep cavity to collapse, e.g., empyema
- Tuberculous or septic infection of the walls of the sinus.
- Constant passage of septic discharges, e g., fæcal or urinary fistula
- Massive growth of fibrous tissue-like cartilage round the sinus.
- Growth of epithelium down the sinus

EXAMINATION OF COMPLICATED SINUSES.—

Inject the sinus with lipiodol, and X ray.

Treatment of Sinus or Fistula.—

- Lay it freely open.
- Find and if possible remove any necrosed bone or other septic bodies.
- Provide efficient drainage or close altogether the cavity to which it leads.
- Scrape away all granulations from its walls
- Swab with pure carbolic in very septic cases.
- Excise the whole track where possible when it is very dense
- Pack the cavity left and allow to heal by granulation.
- Zinc ionization. Ultra-violet light.

SPECIAL TREATMENT OF FISTULA.—

Special plastic operations to close the inner opening, or other special operations.

TREATMENT OF CHRONIC SINUSES, especially those left after opening tuberculous abscesses.—

- Cleanse the sinus with 90 per cent alcohol.
- Inject first with a soft bismuth mixture (bismuth carbonate 1 part to vaseline 2 parts).
- Repeat this once a week.
- Later, when the discharge lessens, use a harder bismuth mixture; e.g., bismuth carbonate 30 parts, white wax 5 parts, paraffin (melting-point 49° C.) 5 parts, vaseline 60 parts.
- The injections can be made at longer and longer intervals if the case is favourable.

The bismuth mixtures are sterilized by placing them in a vessel in water and boiling the latter for an hour. They are injected whilst warm.

There is some reason to think that the bismuth owes its activity in this method of treatment to its capacity for acting as a radio-active substance after exposure to the X rays. Therefore after each injection the X rays should be applied for about fifteen minutes about once a week.

Definition of Ulcer and Ulceration.—An ulcer is a superficial loss of tissue involving a part of the skin or mucous membrane. Ulceration is a molecular necrosis of superficial tissue leading to a loss of substance.

Classified as: (1). Non-specific; (2) Specific; (3) Malignant.

1. NON-SPECIFIC.—
   - *a.* TRAUMATIC—thermal, chemical, mechanical, due to wounds.
   - *b.* PYOGENIC AND PARASITIC.
   - *c.* VASCULAR AND TROPHIC.
2. SPECIFIC, e.g., tubercle, syphilis.
3. MALIGNANT, e.g., rodent ulcer, carcinoma, sarcoma.

General Features of Ulcers.—Points to be noticed:—

1. SURFACE.—

Usually depressed below the healthy surface

Elevated above the surrounding surface in the following: Ulcer with exuberant granulations; malignant or any fungating growth.

Sloughy, smooth, granulating, or fungating, according to the stage of ulceration.

Granulations may be vascular, e.g., in healing ulcers; œdematous, e.g., in tuberculous; fibrous, non-vascular, in syphilitic.

2. EDGE.—

Eroded and dark red or covered with slough: when the ulceration is actively spreading.

Rounded, smooth, thick edge: when the ulcer is stationary.

Shelving, with white, blue, and red zones: healing.

Thick, infiltrated, raised, or everted: new growth.

Clean cut, 'punched out': especially in syphilitic tertiary ulcers.

Gyrate or circinate, i.e., made up of parts of several smaller circles: usually tertiary syphilis.

Undermined, when an ulcer follows a subcutaneous growth, abscess, or gumma: often seen in tubercle, sarcoma, syphilis.

Surrounded by mass of hard epidermis: perforating ulcer.

3. RELATION TO STRUCTURES BENEATH.—

Movable over deep structure: healthy and healing.

Fixed to deep structure: chronic stationary.

Fixed to deep tumour: new growth.

Penetrating deeply into bones and joints: perforating ulcer.

4. DISCHARGE.—

Purulent and free, containing active bacteria: in spreading cases.

Scanty and serous: in healing cases.

Bleeds readily and copiously. malignant.

Bleeds moderately: healthy granulations.

Does not bleed, though granulating: syphilis.

Ulcers—General Features—Discharge, ***continued.***

Leaves chalky deposit: gouty.
May contain special structures, e.g., actinomycosis granules, epitheliomatous cells, etc.

5. CONDITION OF THE SURROUNDING TISSUES.—
Swollen, œdematous, congested: due to vascular causes.
Inflamed: pyogenic ulcers.
Quite healthy, with abrupt transition: tertiary syphilis.
Deep induration: malignant.

6. POSITION.—
Traumatic ulcers may be anywhere, most commonly on exposed situations.
Vascular ulcers: lower half of leg, e.g., varicose ulcer.
Tuberculous ulcers: in neck, groins, or axillæ, i.e., over glands or over joints. May occur anywhere.
Lupus: face (nose, mouth, ears), fingers or toes.
Syphilis (tertiary): may be anywhere, but especially near the knees on lateral surface of upper third of leg (*Fig.* 4), buttocks, outer side of thighs. Also in face, nose, and mouth.
Malignant: anywhere, but most commonly in mouth and tongue, breast, external genitals.
Trophic or Perforating: Site of any old scar or ulcer. Beneath the sole of the foot, usually under the great toe.

7. SHAPE.—
Simple congestive ulcers tend to be round or oval.
Tertiary specific ulcers may be circinate, i.e., formed by the coalescence of several ulcers; or islands of skin may be present in the midst of an ulcer from the same cause.

8. NUMBER.—
Multiple ulcers, especially if widely scattered, indicate some constitutional infection, e.g., tubercle, secondary syphilis
Multiple ulcers all on the same part indicate some local infection by pyogenic bacteria.
Single large ulcers are of either mechanical, congestive, malignant, or tertiary syphilitic origin.

9. RATE OF PROGRESS.—
Short history: rapid healing is found in most simple pyogenic ulcers.
Extreme chronicity: especially in congestive ulcers, also in rodent ulcers.
Steady growth: malignant ulcers.

10. MICROSCOPICAL CHARACTER of the growing edge.—
May show malignant or tuberculous character.

Stages of Simple Ulceration.—

I. EXTENSION.—The inflamed ulcer.
STRUCTURE.—Is that of the wall of an acute abscess.
CHARACTERS.—
Surface: Covered with sloughs or lymph. No granulations.
Discharge: Copious, thin, offensive.
Margins: Acutely inflamed, with well-defined edge.
Base: Thick and adherent.
TREATMENT.—Rest—Fomentations.

II. STATIONARY OR CHRONIC STAGE.—The stage between tissue necrosis and tissue formation, i.e., between sloughing and granulation. Theoretically every ulcer undergoes this stage, but only in chronic ulcers is it well marked. In healthy ulcers tissue regeneration begins directly necrosis is at an end.

STRUCTURE.—The inflammatory zones of hyperæmia and exudation become converted into fibro-cicatricial layers.

The surface is covered by a layer of small round cells, not sufficiently vascularized to form granulations.

CHARACTERS.—

Surface: Smooth, glazed, and shiny.

Discharge: Scanty and serous.

Margins : May be congested, but are not inflamed.

Edges: Rounded, hard, and elevated.

Base: Often adherent to underlying structures, e.g., the periosteum.

Skin around is very congested, and often pigmented, with marked œdema in 'congestive' cases (*Fig.* 5).

CAUSES OF CHRONICITY, i.e., of non-healing of an ulcer.—

1. Defective circulation: in lower parts of legs. In those who stand all day—In old people.
2. Venous obstruction: Varicose veins—After venous thrombosis, e.g., 'white leg'—Pregnancy.
3. Fixation of the ulcer to underlying bone or fascia: Prevents the edges being drawn together—Prevents the base being drawn up to the edges—Prevents the proper vascularization of the base of the ulcer necessary for the formation of granulations.
4. Constitutional conditions: Bright's disease—Gout—Diabetes, etc.
5. Continued irritation: When an ulcer is left dirty and without any dressing.
6. Pressure of œdema. This causes the vessels in the neighbourhood to be occluded, and produces an extension of the ulcer.

VARIETIES OF CHRONIC SIMPLE ULCERS.—

1. Varicose ulcer. Due to congestion of varicose veins of the leg.
2. Congestive ulcer. Due to congestion of the leg from weak circulation, long standing, etc.
3. Eczematous ulcer. Superficial, not deeper than the dermis. Copious discharge.
4. Irritable ulcer. Over the ankle. Small but very painful. Exposes terminal nerve filaments.
5. Ulcer after a burn or other traumatism. When the whole skin thickness is destroyed, and when a large ulcer overlies a dense fascia or bone. Adhesions of the base to this prevent cicatricial contraction.

COMPLICATIONS OF CHRONIC SIMPLE ULCERS.—

Œdema of the parts below, from the pressure of cicatricial contraction on the blood- and lymph-vessels.

Periostitis of the underlying bone, forming a large periosteal node.

Epithelioma: The edges become thickened, everted, or warty—A very slow-growing form of cancer, which may remain for years—Lymphatic extension is very slow, because the lymph channels have been destroyed.

TREATMENT OF CHRONIC SIMPLE ULCERS.—

Posture: Rest, with elevation of the foot.

Ulcers—Chronic Stage—Treatment, ***continued.***

Pressure: Simple or elastic bandages—Starch bandages over dressings—Strapping.

Unna's Paste is the most useful routine treatment, because it combines uniform pressure with the application of a stimulating medicament. It consists of zinc oxide 5 parts, gelatin 5 parts, glycerin 8 parts, boric acid 1 part, and water 6 parts. It is liquefied in a pot placed in hot water and painted over a layer of gauze wrapped round the limb and surrounded by a thin bandage soaked in the mixture. It is changed once a week. In the case of large foul ulcers a window may be cut in the dressings over the sore, and the ulcer dressed daily.

Dressings.—

When the surface is foul: Viscopaste bandages or boracic fomentations, and starch poultices.

For mere indolence: Local application of lotio rubra and ung. scarlet red or crude cod-liver oil dressings.

Improve the local nutrition of the tissues by application of ultraviolet light.

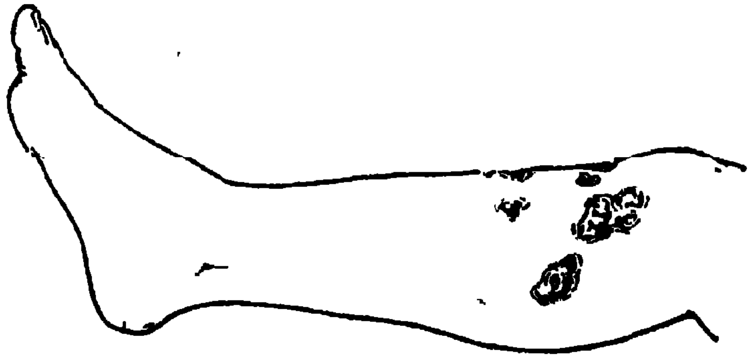

Fig. 4.—Syphilitic ulcer of leg.

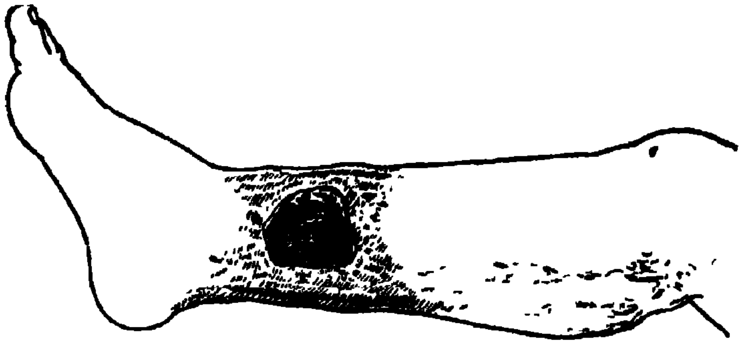

Fig. 5.—Chronic varicose ulcer of leg.

Fig. 6.—Perforating ulcer of foot.

III. STAGE OF REPAIR.—

STRUCTURE.—

The floor is covered with small round cells grouped round new vascular loops, thus forming the granulations.

When the cavity is filled with granulations, the epidermis grows over them from the edge, and so covers the surface.

The granulation tissue becomes converted into fibrous tissue, the deeper layers undergoing this change first.

The contraction of this fibrous tissue is essential to the repair of all large or deep ulcers.

1. It draws the base up to the edges.
2. It draws the edges together.

CHARACTERS.—

Surface: Covered by red granulations.

Edge: Shelves gradually into the ulcer and is on the same level with the granulations. Shows a white, blue, and red margin.

White margin = heaped-up epidermis.
Blue zone = granulations seen through thin, transparent epidermis.
Red zone = granulations.

Base is free from the underlying tissues, i.e., movable over them.

Margins are free from inflammation or congestion.

TREATMENT OF A HEALING SIMPLE ULCER.—

Protection by simple dressing.

Prevent the granulations sprouting up above the skin margins by silver nitrate.

Trophic or Perforating Ulcer of the Foot.—

CAUSES.—

PREDISPOSING.—Diseases of the cord, e.g., tabes or syringomyelia; neuritis, whether diabetic, syphilitic, or alcoholic; peripheral nerve lesions causing anæsthesia.

EXCITING.—Pressure of a corn or an abrasion which is unnoticed and neglected because of the anæsthesia.

CHARACTERS.—The orifice is on the sole of the foot, usually under the ball of the great toe (*Fig.* 6). The margins are formed by heaped-up hard epithelium.

A long sinus leads into the deep tissue of the foot, usually involving carious bones and disorganized joints.

It may have acquired a second opening on the dorsum of the foot (hence the term perforating ulcer).

Probing is conspicuously painless.

TREATMENT.—Rest in bed.—Freely opening up and scraping out the unhealthy tissues. Cutting away all the exuberant epidermis. Subsequent protection from pressure by suitable padding.

Amputation may be necessitated if many of the tarsal bones and joints are diseased.

CHAPTER III

GANGRENE

Definition.—Death of the tissues *en masse* sufficiently large to be obvious to the naked eye.

Usually applied when the whole or a part of a limb or viscus is affected, with all the constituent tissues.

Similar terms but with a restricted meaning: *Necrosis* is the term applied to death—degeneration of a group of cells with the intracellular substance. *Slough* is the dead tissue which becomes separated from the living during the process of ulceration or necrosis.

DIFFERENTIATE *threatened* gangrene from *true* gangrene. They may be similar in appearance, though true gangrene is usually the more severe. Gangrenous tissue never recovers; thus when a patch appears and then disappears it must have been threatened or apparent gangrene, not true gangrene.

Signs.—(1) CESSATION OF CIRCULATION: Loss of pulse—Vessels do not empty on pressure. (2) LOSS OF HEAT. (3) LOSS OF SENSATION. (4) LOSS OF FUNCTION. (5) CHANGE OF COLOUR: Usually dark.

All these signs may be present and yet *recovery may take place* under the following conditions: Small area affected—Absence of the conditions favourable for sepsis—Good circulation in the neighbourhood.

In certain conditions the above signs indicate *inevitable gangrene*:—

When all the vessels going to a part are affected, i.e., when terminal vessels are concerned, e.g., terminal parts of a limb or testis with rotated cord.

When there are conditions favourable for sepsis, e.g., strangulated gut.

Further signs which prove that death has occurred: (6) MUMMIFICATION or drying of the tissues. (7) DECOMPOSITION of the tissues. (8) LINE OF ULCERATION between the dead and living tissues.

Mechanism.—In the final analysis, gangrene is due to one of two main mechanisms:—

1. Death of cells by direct destruction (heat, carbolic acid, some forms of trauma, etc.).
2. Death of cells by reason of failure of blood-supply (senile gangrene, other forms of trauma, etc.).

The second mechanism (2) is by far the commoner and more important. In cases of gangrene search for the cause of the failure of blood-supply.

Varieties.—

1. DRY GANGRENE.—Death followed by *mummification* (*Fig.* 7).

a. PRIMARILY DRY.—Caused by blocking of the arteries whilst the veins remain patent, thus draining the tissues of fluid.

b. **SECONDARILY DRY.**—Caused by a loss of fluids from the tissues by evaporation in a case of moist gangrene which has remained aseptic.

The affected parts become hard, dry, and shrivelled.

Liberated hæmoglobin makes the tissues black.

Liberated fat causes some transparency of the skin, through which the deep tissues can be seen.

Sepsis is either absent, or unable in the absence of moisture to produce decomposition.

Terminates by an ulcerating line of demarcation which separates the mass, chiefly at the expense of the dead tissues.

It has little or no tendency to spread.

If extension occurs it is due to the original cause of the gangrene acting on tissues higher up.

Hence extension is rare, and when it occurs it is usually by leaps, not a gradual, steady process.

Associated with very slight febrile symptoms.

Usually is extremely painful.

Rarely causes death; but death may be hastened by the pain and want of sleep.

2. MOIST GANGRENE.—Gangrene of blood-filled tissues.

Veins as well as arteries are blocked, or congestion occurs simultaneously with arterial blocking.

a. It may become dry (very rarely) if asepsis be maintained until evaporation can take place.

b. It usually leads to rapid *putrefaction*. Hence in the vast majority of cases

Moist gangrene = death + putrefaction.

The affected parts are purple and swollen.

Superficial tissues are raised in blisters.

Deep tissues become emphysematous from the gases of decomposition.

Adjacent living tissues are much inflamed.

Terminates by a broad zone of ulceration which separates the mass at the expense of the living tissues.

It spreads steadily and swiftly by infection and thrombosis of adjacent living tissues.

Associated with marked toxæmic and febrile symptoms.

Pain is great, but is dulled by the toxæmia.

Causes death from septicæmia or secondary hæmorrhage.

The Tissue Changes that follow Gangrene.—These consist in a reaction between the dead and living tissues.

Three factors are concerned:—

1. The living tissues become inflamed, hence need for adequate blood-supply; failure of such blood-supply means further spread of gangrene.
2. The dead tissues are removed partly by molecular and partly by massive necrosis.
3. Bacteria in the dead tissues attack and destroy some of the living tissues.

The actual result will depend on the relation between these three factors, and the dead tissues will suffer one of three fates:—

1. ABSORPTION.—

This really means the disintegration and removal by molecular necrosis of the dead tissue.

Gangrene—Tissue Changes, ***continued.***

The living tissues are healthy and vascular.
Sepsis is absent or insignificant.
The amount of dead tissue is small.
The living tissue produces granulation tissue.
The granulations erode and digest the dead tissue.

2. SEPARATION BY ASEPTIC ULCERATION—
The bulk of the dead tissue is large.
Or the vitality and circulation in the living tissues weak.
The granulating zone cannot penetrate far from its own base.
It merely corrodes the adjacent zone of dead tissue until it is completely separated.
The separated dead mass drops off or remains as a sequestrum.

3. SEPARATION BY SEPTIC ULCERATION.—
The dead tissues contain virulent bacteria.
Acute septic inflammation is caused in the living tissues thereby.
A zone of living tissue undergoes molecular necrosis, i.e., it suppurates
Thus the line of separation between living and dead tissues forms at the expense of the living.
The final demarcation of the living tissue by a granulating zone is distinctly higher up than that of the original gangrene.
The process is one of acute suppuration.
Note.—It is impossible to 'infect' dead tissues. Such tissues may contain the organisms of putrefaction, which do not ordinarily invade living tissue. Surface staphylococci and streptococci are responsible for the active infection of the host tissues.

Varieties of Gangrene in relation to its Cause.—

I. SYMPTOMATIC.—Gangrene caused by some vascular or constitutional condition
(1) Embolic. (2) Senile. (3) Thrombotic. (4) Puerperal (5) Diabetic. (6) Raynaud's. (7) Thrombo-angiitis obliterans. (8) Ergot

II. TRAUMATIC.—
(1) Direct: when an injury kills the part injured. (2) Indirect: when an injury kills a part at a distance from the injury by damage of main vessels.

III. THERMAL AND CHEMICAL—A variety of direct traumatic gangrene
(1) Burns, (2) Frost-bite; (3) Trench foot; (4) Immersion foot; (5) Shelter foot, (6) Carbolic acid

IV. INFECTIVE.—Gangrene resulting from septic inflammation.
(1) Acute inflammatory gangrene. (2) Osteomyelitis. (3) Cancrum oris. (4) Carbuncle. (5) Gas gangrene.

Embolic Gangrene.—

EMBOLUS originates from left auricular appendage in auricular fibrillation from mitral disease or other origin.
An infected embolus can occur from the mitral valves or aortic cusps or wall of the ventricle in infective endocarditis. Rarely a plaque may separate from the wall of an artery diseased by atheroma.

SITE OF IMPACTION OF THE EMBOLUS.—At the bifurcation of vessels, e.g., where the Popliteal divides into anterior and posterior

tibials—Femoral divides into superficial and deep—Brachial divides into radial and ulnar.

SPECIAL FEATURES.—

PAIN: Sudden, severe, and lasting; pallor, paralysis, paræsthesia, and pulselessness.

DRY GANGRENE appears in the extremities, e.g., the toes, especially in asthenic, anæmic people—Affects only the terminal vessels of the blocked trunk—Its extent gives no indication of the position of block—Usually limited by a joint.

MOIST GANGRENE: In a full-blooded patient—Or as a result of secondary venous thrombosis.

DRY AND MOIST GANGRENE is common, e.g., dry gangrene of toes with moist gangrene spreading up the calf.

TREATMENT.—

EMBOLECTOMY.—The site of the embolus can usually be determined, by local tenderness and the distribution of the gangrene and local anatomical factors. Local anæsthesia is used. The vessel above and below is exposed and controlled by clamps. The vessel at the site of embolism is opened and the clot removed, and the vessel walls repaired by fine silk sutures, lubricated by paraffin, and held in finest sewing needles; supplemented by pepsin therapy. The time factor is vital. Embolectomy under six hours from lodgement of embolus is very encouraging. Over twenty-four hours the operation is not worth attempting except as an arteriectomy (partial) to limit spasm.

Senile Gangrene.—

PREDISPOSING CONDITIONS.—Calcareous arteries whose collaterals cannot dilate—Weak circulation—Anæmia or some constitutional disease, e.g., nephritis.

EXCITING CAUSE.—Thrombosis in the arteries, often determined by some injury or inflammation, or cold.

SYMPTOMS.—

Premonitory: Cold feet, numbness, and cramps.
Dry gangrene beginning in one toe, usually the great toe.
With or without inflammation at its origin.
The gangrene has all the signs of the dry variety (*see above*).
Intense pain throughout.

Thrombotic Gangrene apart from traumatism is very rare.
Occurs after typhoid and other long cases of toxæmia.
It is usually in the leg, affecting the femoral.
Dry gangrene follows simple arterial thrombosis usually.

Puerperal Gangrene.—
Occurs after parturition.
Probably is due to thrombosis spreading from the pelvic to the femoral vessels.

Diabetic Gangrene.—Three distinct factors in its causation:—

1. A BLOOD CONDITION favourable to septic processes.
2. AN OBLITERATING ARTERITIS, especially in the tibials.
3. NEURITIS, with some posterior sclerosis of the cord.

Diabetic Gangrene, ***continued.***

MOIST GANGRENE in its worst form is the common type.

Elderly patients—Chronic diabetes—Begins as a whitlow or suppurating corn—Rapidly spreads and is very fatal—Is very likely to give rise to diabetic coma.

DRY GANGRENE usually results from arterial sclerosis and neuritis—It is much less painful than other forms of dry gangrene—When neuritis is a chief cause it may be quite anæsthetic.

The insulin treatment of diabetes has made some modifications. Formerly the gangrene was commonly moist, now it is commonly dry. Insulin has not greatly lessened the incidence of gangrene but has reduced its mortality. Statistics are difficult as diabetics survive longer (due to insulin), and the arterial disease is more common.

Raynaud's Disease.—

Young patients—Generally women.

Associated with angioneurotic œdema, paroxysmal hæmoglobinuria, and other neuroses.

Affects fingers, toes, ears, or nose, in superficial patches symmetrically on both sides of the body.

Of slow development, producing anæmia, cyanosis, and then a superficial slough.

Very painful.

TREATMENT.—In the pregangrenous stages, i.e., the stage of vasospasm, much relief can be afforded by sympathectomy. Opinions are divided as to the best method, but stellate (cervico-dorsal) ganglionectomy is the method most widely performed. Late relapses are not unknown.

Thrombo-angiitis Obliterans.—

Usually in the lower limbs.

Arterial thrombosis followed later by venous thrombosis.

Before the onset of gangrene, intermittent claudication and various sensory changes occur.

Treat by excision of the lumbar sympathetic ganglia, L. 2–5.

Padutin therapy lessens arterial spasm and may be of use in early cases.

Direct Traumatic Gangrene.—

The parts which die are at the seat of injury.

The gangrene is caused by a lesion of the blood-vessels locally, thrombosis, obliteration by pressure or rupture.

Trophic disturbances and sepsis may take a large part in the causation.

1. CRUSHED LIMBS.—(*See* CONTUSED WOUNDS.)
2. SPLINT PRESSURE.—Too tight bandaging or ill-fitting or ill-padded splint—Usually over a bony prominence—Caused by a pressure-obliteration of vessels—Affects the superficial tissues over a bone.
3. BED-SORES occur over bony points on which a bedridden patient lies—Any prominent vertebral spine—Sacrum or posterior iliac spines—Trochanter—Heels.

   Especially in condition of emaciation—Debility of fevers, e.g., enteric—Any spinal cord lesion, especially traumatic paraplegia.

   TROPHIC DISTURBANCE is the most important factor in spinal cases. The sores are then apt to be acute in their origin and progress.

   IRRITATING DISCHARGES—e.g., urine and fæces—also often play an important part in the causation.

CONDITION.—Superficial skin slough, limited to points of pressure in most cases—Deep spreading sloughing in acute cases with spinal cord lesions.

PREVENTION.—Careful nursing, i.e., Smooth clean sheets—Constant cleansing from urine or fæces—Keeping the back as dry as possible (washing with spirit after soap and water, dusting with boracic powder)—Use of water bed or air cushions to prevent localized pressure on the prominent bony points.

TREATMENT.—General cleanliness.
Stimulating ointments, e.g., resin or Friar's balsam.

4. ACTION OF CORROSIVE CHEMICALS.—Strong acids, e.g., sulphuric or nitric acids, carbolic acid—Strong alkalis, e.g., potash.

Course, varieties, and treatment are the same as in burns; but with acids and alkalis neutralization should be attempted if the injury is quite recent, so as to stop the corrosive action spreading.

Indirect Traumatic Gangrene.—Caused by an injury obstructing the vessels. The parts that die are at a distance from the injury.

1. LIGATURE OR THROMBOSIS OF MAIN ARTERY when the circulation is too weak or the vessels are too diseased to carry on the collateral circulation.

Similar to embolic gangrene.

2. OBSTRUCTION OF BOTH THE MAIN ARTERY AND VEIN.—By ligature or injury—By tight bandaging—By strangulation of an internal organ—Primary rupture of an artery and secondary pressure on veins.

Moist gangrene occurs up to the level of adequate collateral circulation.

The incidence of gangrene is governed by the effectiveness of the collateral circulation. Gangrene less frequent if main vein is ligated as well as the artery.

Infective Gangrene.—Caused by the direct toxic effect of micro-organisms.

1. LOCALIZED GANGRENE.—Usually staphylococcal. Here a dense condition of the tissues, e.g., dense bone or subcutaneous fascia, determines the gangrene because the vessels become strangulated by inflammatory exudation in an unyielding matrix.

a. NECROSIS OF BONE.

b. CARBUNCLE OR BOIL OF THE SKIN.

2. SPREADING GANGRENE.—

a CANCRUM ORIS AND NOMA.—One attacks the mouth, the other the genitals—Affects weakly children after exanthemata—Chiefly streptococcal—Slough begins on the mucous membrane, and spreads right through to the skin—Destroys the whole cheek down to the bone.

Produces: sapræmia, septicæmia, or septic pneumonia.

Treatment.—Cut away all the affected parts with the diathermy or actual cautery.
Rub pure carbolic or nitric acid into raw surfaces.
Antistreptococcal serum.
Subsequent plastic operation.

b. GAS GANGRENE, or Malignant œdema, or Acute emphysematous gangrene, or Acute traumatic gangrene.

Exciting cause: Large lacerated wound with much dirt contamination, or virulent infection of a small wound.

Gas Gangrene, *continued.*

Organisms.—i. Bacillus of malignant œdema.
ii. *B. aerogenes capsulatus.*
iii. *B. œdematiens.*

Usually mixed with other bacteria.

All are anaerobic.

All have intense peptonizing action and produce gas by their growth. They are commonly found in garden soil. The anaerobic bacteria spread rapidly in the sheath of the muscles, and for a time the infection is localized in one or more of these structures.

Signs.—Occur within 24 hours of infection.

Rapidly spreading acute cellulitis—Parts become dark coloured—Surgical emphysema is well marked—Sloughing only occurs if the patient lives long enough. Characteristic foul odour.

Symptoms.—Acute septicæmia—Slight rise of temperature, later becoming subnormal—Mental state usually quite clear.

Treatment.—Multiple incisions into swollen tissues. Excision of the infected wound and of muscles specially involved. High amputation if the above fail to check the spread of infection. Polyvalent anti-gas-gangrene serum. Chemotherapy, e.g., sulphapyridine intravenously, then orally. Deep X-ray therapy according to Kelly's technique.

Treatment of Gangrene.—

1. RENDER THE PARTS AS ASEPTIC as possible.
   Spirit and biniodide of mercury 1–500 are specially valuable because the spirit dries and hardens the skin.
2. KEEP THE PARTS DRY and encourage evaporation.
   This maintains a dry type of gangrene, or rarely
   Converts a moist into a dry gangrene.
   Dry sterilized dressings applied with all aseptic care
   HOT FOMENTATIONS should be used with great caution—
      Never in cases of dry or aseptic gangrene,
      But only when sepsis is obviously present in moist gangrene.
      A fomentation is the surest way of converting a dry aseptic into a moist septic gangrene.
3. ELEVATION OF THE PARTS, KEEPING THEM WARM.—
   To encourage the circulation.
   To empty the veins, and thus prevent the moist type of gangrene.
   To prevent the spread of the gangrene in cases where the collateral circulation is very feeble.

d. AMPUTATION.—In all cases except, perhaps—
   a. Senile gangrene with marked calcareous arteries and feeble general condition or albuminuria.
   b. Diabetic gangrene with marked acetonuria (acidosis), or commencing coma.
   c. Acute infective gangrene which has involved the trunk, or in which septicæmia has already developed.

   WHEN AND WHERE TO AMPUTATE must be determined by
      a. The nature of the gangrene, dry or moist.
      b. The presence and virulence of septic processes.
      c. The condition of the main blood-vessels.
      d. The general condition of the patient.

When to Amputate (i.e., at what period)?

In dry gangrene: Directly a definite line of demarcation appears, or directly the final death of the part is inevitable—When pain is becoming intolerable.

In moist gangrene: Directly active septic processes or a line of ulceration are evident.

Where to Amputate?

With diseased vessels: High amputation, e.g., lower third of thigh for senile gangrene of toes.

With active sepsis but healthy vessels: Amputation well above the inflamed area, e.g., middle of calf for gangrene of foot.

With healthy vessels and no sepsis: Amputation through the line of demarcation, or just above it.

BURNS

Burns and Scalds.—Give rise to inflammation, ulceration, or gangrene.

DEGREES OF INJURY produced by burns or scalds.—

1. Burns involving partial skin loss.
2. Burns causing total skin loss.

STAGES IN THE COURSE OF A BURN.—

1st Stage: Shock.—At the time of injury: Severe (*a*) in proportion to the extent of the surface; (*b*) In children; (*c*) In burns of trunk, especially abdomen.

When the shock is fatal, the internal organs, and especially the portal system, are found engorged, as though from vasomotor paralysis.

2nd Stage: Inflammation.—During the separation of dead tissues. Lasts one or two weeks, in proportion to the depth, extent, and septic infection of the burn. Any variety of septic complication may ensue—Gastro-intestinal inflammation, ulceration may result from septic absorption—Liver necrosis may occur due to toxæmia.

3rd Stage. Healing.—From time of separation of dead tissues until recovery—Will vary according to the degree.

CAUSES OF DEATH.—Shock—Gastro-intestinal inflammation and ulceration—Liver necrosis—Septic absorption—Pneumonia.

TREATMENT.—

For Extensive Burns with Much Shock.—Cases presenting much shock, especially children, should be treated by hot air. A cradle is placed over the patient and heated by electric lamps or hot bottles, the whole being covered by blankets. Warm sweetened drinks. Plasma transfusion. Treat the shock before the burn. Morphine.

In Partial Skin-loss Burns.—Clean wound, apply gentian violet jelly, triple dye jelly, or vaseline gauze. Do not apply tannic acid preparations. Blisters should be cut and dusted with sulphanilamide powder.

In Deeper Burns.—Render aseptic (under anæsthetic if necessary). Dress with sulphanilamide powder and cover with 'tulle gras'.

Large wounds of the trunk can be treated in an irrigation envelope. Milton should be circulated over the burnt area three times a day. Sepsis must be eliminated at all cost. All deep burns must be skin-grafted as soon as possible.

Electrical Burns are always deep and should be excised and skin-grafted.

FROST-BITE, TRENCH FOOT, SHELTER FOOT, AND IMMERSION FOOT

Frost-bite.—

CAUSES.—(1) Direct effect of cold on exposed parts; (2) Pressure of inflammatory exudation caused by too rapid thawing.

CHARACTERS.—

a. SUPERFICIAL DRY GANGRENE of nose, ears, fingers, or toes—The slough which separates is superficial—The process is painless—It occurs as the direct effect of the cold.

b. MOIST GANGRENE of an inflammatory character from too rapid thawing—Is extremely painful—Often ends in superficial ulceration rather than actual gangrene.

TREATMENT.—Very gentle gradual thawing—Elevation of part, with gentle bandaging, to prevent congestion—Keep part aseptic, dry, and elevated when gangrene has occurred.—Restrict fluid intake and give diuretics to limit exudation into the tissues.—Later, amputation of toes may be required.

Trench Foot is caused by continual standing in cold wet stockings or socks. Common in the war of 1914–18. The foot becomes swollen, cold, and painful.

TREATMENT consists in very gradually warming the foot, and this should be undertaken in hospital.

Shelter Foot occurs in people who spend nights in cold, damp shelters with their legs dependent over the sharp edge of the wooden seat. There is often a deficiency of C and P vitamins with an increase of capillary permeability. The condition may go on to gangrene.

Immersion Foot has been quite common in shipwrecked people who have spent a long time in waterlogged boats or rafts. Increased capillary permeability with œdema of the feet occur, passing on to gangrene.

TREATMENT consists in keeping the feet cold and very, very gradually warming the œdematous tissues. Sepsis must be prevented at all costs. Amputation is required for the severe cases.

SKIN-GRAFTING

Uses.—(1) To facilitate healing of any large superficial wound when much skin is destroyed, or (2) Any large ulcer with great loss of skin; (3) When as the result of a deep burn a wound is left which can only heal by great contraction.

Methods.—

1. REVERDIN'S.—Minute fragments of epidermis only. Only of limited application.
2. STAIGE-DAVIS'S.—Small deep grafts or pinch grafts. Useful in covering in large areas. Should not be used on hands or face.
3. THIERSCH'S.—Thin razor grafts. Large pieces of epidermis with some dermis.
   Is used when large areas have to be covered, e.g., after amputation of the breast, in closing a large burn.

Preparation of the Wound.—It must have no active sepsis or irritating discharges—It must be recent and vascular—Exuberant granulations must be scraped away, but small recent vascular granulations may be left—Bleeding must be stopped by gauze pressure over protective while grafts are cut and frosted with sulphanilamide powder.

Preparation of Grafts.—Skin must be rendered aseptic—Cut grafts with flat, sharp razor—Hold skin flat and tense with strips of wood—Keep razor surface wet with water all the time—Cut the grafts as large as possible—Best taken from the thigh. Padgett dermatome may be used for cutting the grafts.

The cut should be down into the fibrous vascular layer of the dermis, but not through it.

The graft beds are dressed with simple ointment, and heal quickly, because the deep layers of skin regenerate those taken away.

Laying the Grafts.—Grafts have been placed in warm saline fluid—Laid on the wound absolutely flat—Wound surface should be completely covered—Dressed with wide-meshed gauze impregnated with medicated paraffin wax (tulle gras)—(Slight pressure is necessary to prevent the graft floating off)—Use no chemical antiseptics throughout.

4. WHOLE THICKNESS OF THE SKIN.—In covering small wounds, e.g., eyelids, or any part of the face, e.g., after the removal of rodent ulcers.

Thin hairless skin should be chosen, e.g., prepuce, parts of the outside of the arm; or a pedicled flap may be taken from the chest (*Fig.* 8).

The graft is cut a little larger than required—Is sewn accurately to the edges of the gap—Firm pressure is needful afterwards. If graft has a pedicle, the latter is cut on the eighth day.

This method gives a more satisfactory result than Thiersch's, because the graft consists of the whole skin thickness and is thicker and tougher.

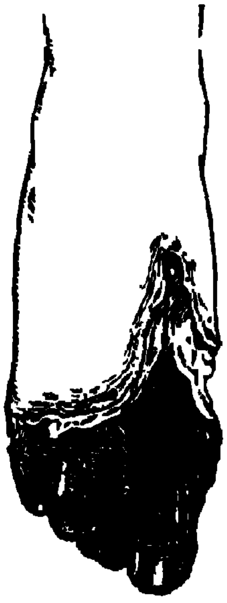

Fig. 7.—Dry gangrene of foot, showing line of demarcation.

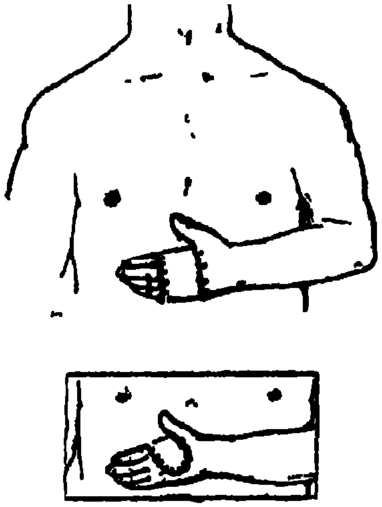

Fig. 8.—Skin-grafting by pedicled flap.

Skin-Grafting, *continued.*

5. TUBULAR GRAFTS.—The whole thickness of the skin with the subcutaneous tissue is dissected up as a flap, remaining attached at one end. The flap is then sewn together as a tube and the free end of the tube is implanted into the edge of the area which it has to cover finally. After a week or more, the base of the tube is cut from its original site. Lastly, it is unfolded so as to convert the tube once more into a flap and then spread over the raw area it has to cover and sutured in place.

In this way, when the face has been extensively burned, the whole skin can be replaced by one or more flaps from the chest; or the skin of the thigh may be brought down to cover the ankle (*Fig.* 9).

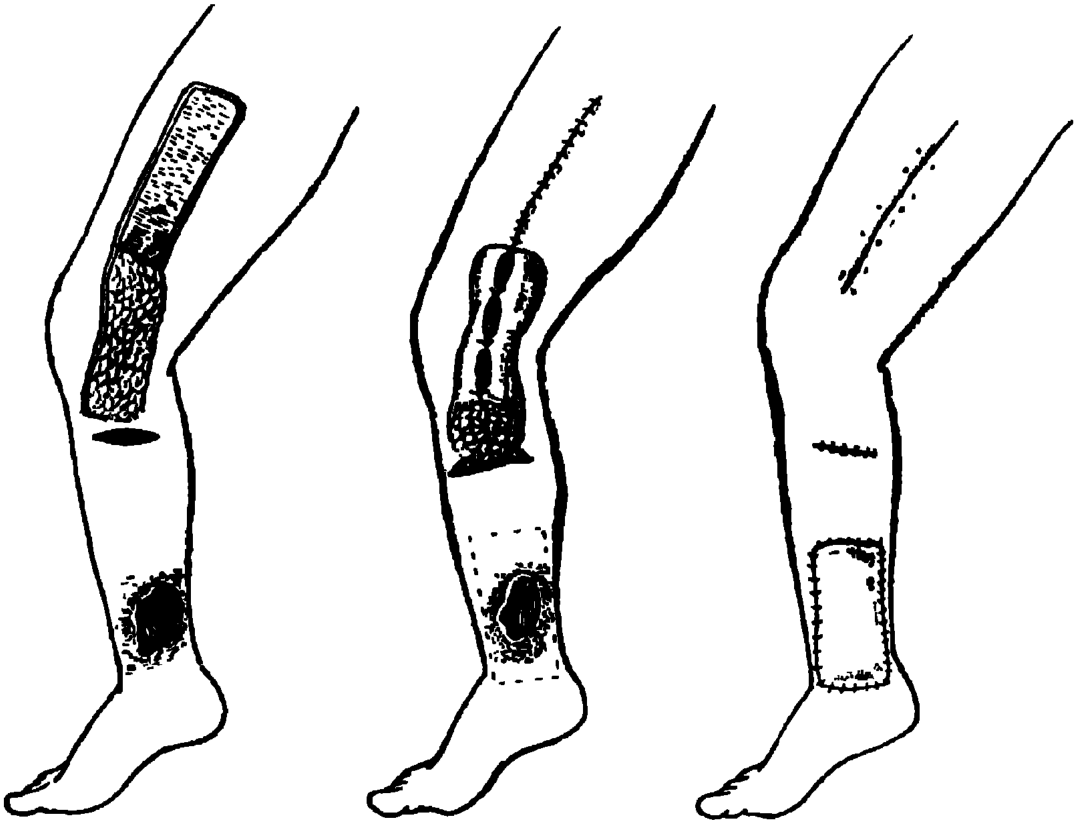

Fig. 9.—Tubular skin-grafting. A long flap is taken from the thigh with pedicle below. It is turned down and its free end sewn into the middle of the leg. Its edges are sewn together to form a tube. Ten days later, the original pedicle is cut and the graft turned down and sewn over the raw area at the ankle. Later the secondary pedicle is cut away.

CHAPTER IV

ACUTE SURGICAL INFECTIONS.

CELLULITIS

Definition.—Septic inflammation of subcutaneous and fascial connective tissue.

Causes.—

Injury, or wound, or abrasion of skin or mucous membrane—especially deep wounds with small external openings.

BACTERIA.—Staphylococci (*albus* and *aureus*) (slight cases), streptococci (severe cases), or bacillus of malignant œdema (emphysematous gangrene).

PREDISPOSING CAUSES.—Albuminuria, diabetes, debility.

Symptoms.—

INCUBATION PERIOD of about two or three days, during which there is some general malaise.

ONSET.—Usually with a sharp rise of temperature and rigor—Rarely a subnormal temperature occurs, and this in the worst cases.

CONSTITUTIONALLY.—All symptoms of fever, with great pain and sleeplessness—Delirium in very bad cases. There may be large subcutaneous abscesses (staphylococcus), or a spreading sloughing of the cellular tissue (streptococcus). The tendency is to spread up the limb along the lines of the fascial planes rather than to burst through the skin.

LOCALLY all the signs of inflammation occur: Tenderness, redness, brawny induration and œdema—Intense throbbing pain—The outline and margin are very indefinite and ill-defined.

COMPLICATIONS.—Some form of serositis by extension, e.g., Meningitis from disease in the scalp or orbit—Pericarditis, Pleurisy, Mediastinitis, from cellulitis of the neck—Peritonitis from pelvic cellulitis—Septicæmia or Pyæmia from general infection.

Treatment.—

Early stages: warmth and rest to the affected part, and administration of sulphonamides. Free action of bowels and kidneys. Attention to general body health.

If pus is suspected, free incisions along the axis of the limb down to the deep fascia and packing incisions lightly with gauze soaked in hypertonic saline (5 per cent). When bleeding from incisions has ceased, routine hot eusol baths at 4-hourly intervals to the affected part.

Special Forms.—

SCALP.—The affected parts are bounded by the occipito-frontalis aponeurosis, i.e., by the superciliary ridges in front, superior curved lines of the occiput behind, the temporal ridges at the side.

The whole scalp over this area may become lifted up by a bag of pus. *Meningitis* or necrosis of the skull may follow.

TREATMENT.—Free incisions and drainage.

Cellulitis—Special Forms, ***continued.***

ORBIT.—Caused by penetrating wounds or extension from boils on the eyelids.

RAPID COURSE.—Very great pain, with high temperature and delirium. The eyeball is pushed frowards and vision lost.

COMPLICATIONS.—Meningitis, septic thrombosis of the cavernous sinus, suppurative panophthalmitis, optic neuritis. Later complications are: destruction of the ocular muscles, and retraction of eyelids.

TREATMENT.—Free incisions and fomentations. The eyeball must be sacrificed by evisceration in severe cases or where panophthalmitis supervenes.

CERVICAL OR SUBMAXILLARY CELLULITIS (LUDWIG'S ANGINA). —Affects the deep cervical fascia, usually beginning in the submaxillary region and spreading rapidly downwards and inwards.

CAUSED by an intrabuccal or pharyngeal infection, usually in old and debilitated people.

RAPID SLOUGHING and suppuration occur in the plane of the deep fascia, at the root of the tongue, and spreading down the pretracheal fascia to the superior mediastinum and pericardium, or down the prevertebral fascia to the posterior mediastinum and pleura.

COMPLICATIONS.—Œdema glottidis—Sublingual abscess—Suppurative mediastinitis—Pericarditis—Empyema.

TREATMENT.—Free incisions, especially in the mid-line above the hyoid bone—Tracheotomy for œdema glottidis.

PELVIC CELLULITIS.—

CAUSES.—Septic conditions of the uterus, lacerated cervix, punctured wounds of the rectum and bladder. Also due to diseases of bladder, vesicles, and prostate in the male.

SIGNS.—Constitutional signs are severe. The uterus is pushed to one side, and a phlegmon arises on one side of the pelvis and spreads up the round ligament or cord to the inguinal region. An abscess forms either in the pelvis or in the inguinal region.

DIAGNOSIS.—An inguinal abscess of this kind has to be distinguished from: (1) Bubo, or other lymphatic swellings; (2) Inflamed hernia; (3) Psoas abscess, (4) Appendicitis.

COMPLICATIONS.—Abscess may burst into vagina, rectum, bladder, or intestine.

TREATMENT.—Hot douches and fomentations. Open through the vagina or the inguinal region if abscess formation occurs.

Infections of the Hand.—

INFECTION OF TERMINAL PULP SPACE OF FINGER.—

Pus collects in the connective-tissue space, and by its tension deprives the bone of blood-supply.

If unrelieved, the result is necrosis of the ungual phalanx; but the epiphysis does not suffer.

TREATMENT.—Incision on side of pulp of finger must not extend within ¼ in. of terminal flexor crease, otherwise flexor tendon sheath will be opened.

PARONYCHIA.—Septic infection of the base of the nail-bed.

Begins at one side and spreads to the other.

TREATMENT.—Lateral incisions, removing part or the whole of the base of the nail (*Fig.* 10).

WHITLOW.—This term has been loosely applied to various infective inflammations of the fingers, and should be replaced by the more definite entities described below.

SUBCUTICULAR OR SUBEPITHELIAL INFECTION.—A localized inflammation causing a serous and then purulent bleb beneath the epidermis.

LYMPHANGITIS.—Usually a streptococcal infection through a minute wound or abrasion.

Bright-red blush, followed by pitting œdema.

Spreads rapidly up the arm, first by red streaks.

Glands at the elbow and axilla become enlarged.

Fingers can move; no special tenderness over tendon sheaths.

May present several degrees, viz.. (1) A localized fugitive process; (2) A localized process with extension into deep planes (cellulitis) or tendon sheaths; (3) A rapidly fatal septicæmia.

Treatment.—Treat the wound. Fomentations and rest and chemotherapy, avoiding incisions unless local suppuration develops. Flat elastic band above elbow to promote hyperæmia and to localize infection.

CELLULITIS OR SUBCUTANEOUS WHITLOW.—May be streptococcal or staphylococcal.

Begins like lymphangitis.

Causes suppuration under deep fascia, with sloughing of latter.

Dark-red, brawny, indurated swelling chiefly at the back of the hand and forearm.

Treatment.—Multiple incisions and fomentations.

TENOSYNOVITIS.—Infection of tendon sheaths, usually of the flexor tendons.

Anatomy (*Figs.* 12, 13, 14).—

- The three middle digits have sheaths only as far as the front of the knuckle-joints.
- The little-finger sheath usually extends into a large sheath in the hand which goes above annular ligament and is known as the ulnar bursa..
- The thumb flexor sheath extends similarly into a separate sheath in the hand and above the wrist known as the radial bursa.
- The ulnar and radial bursæ frequently communicate.

Signs.—

- Exquisite tenderness to pressure over affected sheath.
- Finger is rigidly flexed.
- Great pain on passive extension.

Extension of Infection.—

- Thumb and little finger: Infection spreads into radial or ulnar bursa, and thence above into forearm deep to profundus tendons.
- Three middle digits: Infection spreads into one of the fascial spaces in the palm.

Treatment.—

- Open over point of maximum tenderness and swelling.
- Make lateral incisions on front of fingers between the joints.
- Open ulnar or radial bursa by incision through the palm, as indicated in diagram (*Fig.* 11).

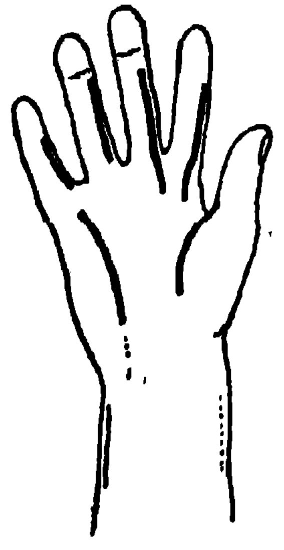

Fig. 10.—Incisions employed in paronychia and upon the dorsum of the hand in fascial space infection. That on radial side of index metacarpal is for the thenar space. Those in the third and fourth clefts are for the mid-palmar space.

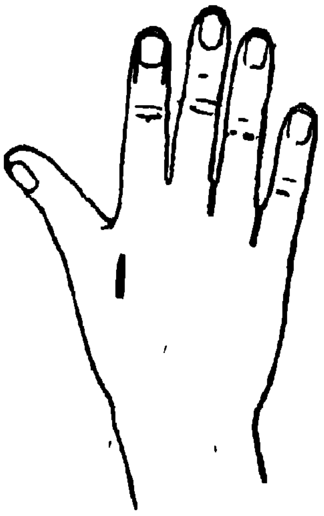

Fig. 11.—Treatment of tenosynovitis of the fingers. Lines showing area of possible incisions for infections of the various tendon sheaths. (In case of doubt the free incision of the whole sheath is to be advised.)

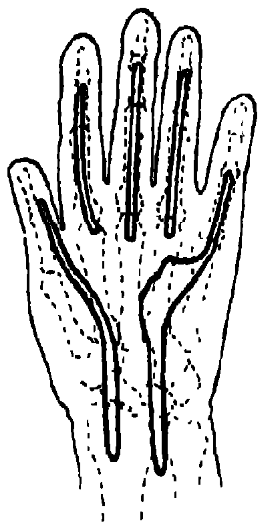

Fig. 12.—Diagram to show flexor tendon sheaths.

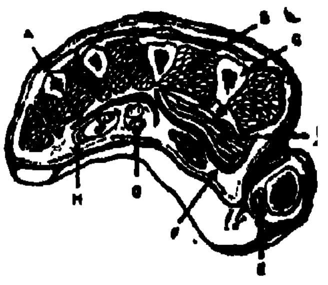

Fig. 13.—Cross-section of hand 3·5 cm. proximal to the metacarpo-phalangeal joint. A, Middle palmar space; B, Interosseous muscle; C, Metacarpal; D, Adductor pollicis; E, Flexor pollicis; F, Thenar space; G, Finger flexors; H, Ulnar bursa.

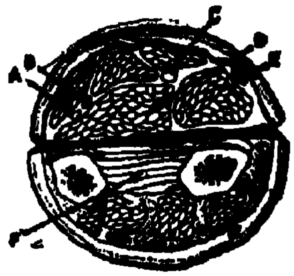

Fig. 14.—Cross-section of forearm 7 cm. above radial styloid, showing incision made transversely in juxtaposition to ulna and radius through anterior interosseous space, demonstrating that an incision can be made here and not injure important vessels and nerves. Note tissue between radial artery and line of incision. A, B, Radial artery and nerve; C, Median nerve; D, Flexor tendons; E, Ulnar artery; F, Pronator quadratus.

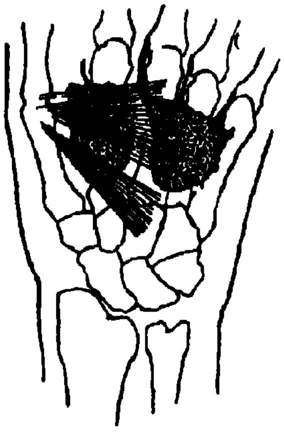

Fig. 15.—Thenar and mid-palmar fascial spaces injected with bismuth. The cross-shading is the adductor pollicis. Note the extension of the spaces along the tendons of the lumbrical muscles.

(*Figs.* 10–15 *after Kanavel.*)

Open forearm suppuration by lateral incisions opening the space deep to the profundus tendons (*Fig.* 14).

After opening and decompressing all pus under tension, put the affected part at rest by immobilizing it and elevate the limb. Give chemotherapy. Radiant heat and short-wave diathermy are valuable therapeutic agents and replace treatment with hot arm baths and moist applications.

RESULTS.—Very liable to be followed by sloughing of the tendons.

FASCIAL SPACE INFECTION.—There are two main loose fascial spaces in the hand, viz.: (1) Thenar space: In the thumb muscles, extending into palm as far as middle metacarpal and containing the flexor tendon and lumbrical muscle of the index finger. (2) Middle palmar space: Beneath the main flexor tendons and the palmar fascia (*Figs.* 13, 15).

Each of these spaces is easily infected from the tendon sheaths, the thenar space from the thumb or index finger, the mid-palmar space from the middle, ring, or little finger (*Fig.* 14).

Each space extends along the corresponding lumbrical muscle to the interdigital clefts.

THENAR SPACE ABSCESS.—Spreads up radial side of index finger or between index and middle fingers.

Open by incision on dorsum, on radial side of neck of index metacarpal (*Fig.* 10).

MIDDLE PALMAR SPACE INFECTION.—May spread up the lumbricals to clefts between middle, ring, and little fingers.

Open by incision in third and fourth clefts (*Fig.* 10).

ERYSIPELAS

Definition.—A very contagious disease; consisting of a spreading inflammation of the skin due to infection by one of the *Streptococcus pyogenes* group.

Causes.—Predisposing: (1) A wound or abrasion; (2) Constitutional debility; (3) Bad hygiene.

Pathology.—The lymphatics of the skin in the margin are crowded with chains of streptococci. In the regions where the disease has come and gone there is marked leucocytosis but no cocci.

Symptoms.—Malaise with a rigor and headache.

RASH appears within twenty-four hours. It appears first round the wound, which breaks open. It is of a vivid red colour, which fades on pressure. Pain and swelling are not much marked. The eyelids and scrotum when affected become very œdematous. Vesicles and bullæ form superficially, and a fine desquamation occurs, with some staining of the skin as the rash fades away.

THE MARGIN is well marked, rather gyrate, slightly swollen, rapidly advancing.

Sloughing of the skin rarely occurs, and then usually in cases of scrotal affection.

LYMPHATIC GLANDS in the neighbourhood are enlarged and tender.

EXTENSION may occur by the lymphatics or veins to the deep structures, or pyæmia may be set up.

CONSTITUTIONALLY.—Patient is very ill, with high temperature—102°–104°. Delirium is frequent, especially when the scalp is affected.

COURSE.—Tends to spontaneous recovery in one to three weeks.

Erysipelas, *continued.*

Varieties.—

FACIAL ÉRYSIPELAS is often apparently idiopathic and recurrent. It is accompanied by great œdema. It is liable to be complicated by meningitis.

FAUCIAL ERYSIPELAS.—Spreads from the exterior to the pharynx Causes great swelling of the parts, with a tendency to œdema glottidis. Sloughing or ulceration may follow. Massive enlargement of the glands at the angle of the jaw.

SCROTAL ERYSIPELAS.—Causes great œdema, and in children a tendency to sloughing.

CELLULO-CUTANEOUS ERYSIPELAS partakes of the character of both cellulitis and erysipelas, affecting the skin and subcutaneous tissue The margin is less sharply defined; the tendency to general septic infection and sloughing of the skin is greater than in either of the simpler diseases.

Diagnosis.—

THE EXANTHEMATA, especially scarlet fever. In these the rash appears at the same time in different parts of the body The rash has specific characters, and there are other symptoms, e.g., coryza or tonsillitis.

CELLULITIS.—There is more swelling and œdema, and the margin is not well defined.

DIFFUSE ERYTHEMA NODOSUM.—Occurs generally on the legs, with few or slight febrile symptoms. Isolated and ill-defined nodules are its chief feature.

ERYTHEMA SOLARE (sun-burn) is limited to the parts exposed, it does not spread, and there is but little fever.

ACUTE ECZEMA —There is a copious exudation, and little tendency to pass away.

Effect on other Lesions.—Chronic ulcers, simple, tuberculous, or specific, may rapidly heal. Sarcomata have disappeared after erysipelas, this providing the basis for Coley's treatment of the former.

Treatment.—

STRICT ISOLATION, especially from surgical patients.

DRESSINGS of saturated solution of MAGNESIUM SULPHATE to relieve pain and limit the diffusion of the virus.

Painting with 40 per cent aqueous solution of ICHTHYOL after scarification.

Painting the healthy skin beyond the rash with irritants, e.g., IODINE or BRILLIANT GREEN, or scarification, to produce leucocytosis.

SULPHONAMIDE therapy, orally or intravenously.

ANTISTREPTOCOCCUS SERUM, 20–30 c.c. given subcutaneously twice a day in bad cases.

WOUND INFECTION

Causation.—An infection by the ordinary pyogenic bacteria, which are derived from the skin or mucous membrane of the patient, or by contact with infected materials or instruments.

Local Signs.—The wound becomes inflamed, i.e., painful with red and swollen edges. Cellulitis may spread to neighbouring structures. When a stitch is the infective agent, the signs may be later and less manifest, and appear round the stitch-hole.

Local Treatment.—Removal of stitches, and hot fomentations. Daily irrigation with peroxide solutions.

Constitutional Signs of Infection.—These may be of five different grades according to the amount and character of the toxic agent: (1) SEPTIC TRAUMATIC FEVER; (2) HECTIC; (3) SAPRÆMIA; (4) SEPTICÆMIA; (5) PYÆMIA.

Septic Traumatic Fever is the fever which accompanies an ordinary septic wound. It is most marked when the wound is closed. The temperature rises to 101°–103°, often with a rigor. It falls rapidly when the wound is opened, and is at an end when the layer of granulations prevents further absorption.

Hectic Fever is caused by the CONSTANT ABSORPTION OF SMALL DOSES OF TOXINS. Usually the temperature rises to 99°–101° every evening. It accompanies chronic suppuration, especially that of septic tuberculous foci.

Sapræmia is caused by the absorption of a LARGE DOSE OF TOXIN. A large or virulent infective focus exists (e.g., a septic placental remnant in utero, or a localized appendicitis), and from this toxins (but not bacteria) enter the circulation.

SYMPTOMS.—

A RIGOR, with rapid rise of temperature to about 104°, is the first symptom. In the worst cases the temperature remains subnormal.

VOMITING and DIARRHŒA, delirium or coma, dyspnœa, or albuminuria, may occur, according to the organs which react most to the toxins.

THE USUAL SIGNS OF SEVERE FEVER—dry tongue, quick pulse, scanty urine, and headache—are the rule.

All the symptoms subside rapidly when the infective focus is freely opened and cleared out.

TREATMENT must be directed to the cause. Also hypodermic, venous, or rectal saline transfusions are of the utmost value in diluting and excreting the toxins. Chemotherapy.

Septicæmia is caused by the absorption and development of bacteria in the blood of the patient.

BACTERIOLOGY.—

Streptococcus pyogenes is the cause in more than half the cases, and nearly all cases of puerperal origin, and most of those of ulcerative endocarditis.

Pneumococcus and *Staphylococcus* are next in frequency. *Bacillus coli*, *B. œdematis maligni*, *B. pyocyaneus*, and *Gonococcus* are rarer causative agents.

PREDISPOSING CAUSES.—

Special virulence of the bacteria, e.g., in small post-mortem wounds—Special debility of the patient—Large lacerated wound area.

SYMPTOMS.—

LOCAL INFLAMMATORY signs may be of any degree.

TEMPERATURE rises to 104°–105° with a rigor, and remains fairly high without marked intermissions. The temperature becomes subnormal, whilst the pulse rises, in fatal cases.

Septicæmia—Symptoms, *continued.*

The symptoms are the same as those of sapræmia, but of a further intensity.

Blood-stained diarrhœa, blood and albumin in the urine, petechiæ in the skin, and rapid coma are all very significant.

COURSE.—It is usually fatal in five to seven days.

POST-MORTEM SIGNS.—

These are mostly due to early decomposition and to HÆMOLYSIS, i.e., solution of the red corpuscles. Coagulation and rigor mortis are ill marked. The blood-serum is lakey. Hæmorrhages are seen in the intima of vessels and heart. The serous cavities contain blood-stained exudation. The viscera, especially the spleen and lungs, are soft, pulpy, and congested, and their epithelium has undergone CLOUDY SWELLING.

Similar phenomena are seen after death from all acute infectious diseases, also in sapræmia, pyæmia.

DIAGNOSIS.—

IN SEPTICÆMIA organisms are found in the blood.

IN SAPRÆMIA AND SEPTIC TRAUMATIC FEVER: (1) The presence of an inflamed wound or local infective focus in which the local signs are in proportion to the constitutional; (2) The rapid amelioration of symptoms on freely opening the infective focus; (3) The fact that repeated blood examinations give a negative bacterial result.

HYPERACUTE EXANTHEMATA, in which the patient dies before the rash appears, are practically cases of acute septicæmia, indistinguishable from those of traumatic origin.

PYÆMIA is distinguished by the repeated rigors, the intermittent temperature, and local abscesses.

TREATMENT.—

Local treatment on general lines must be undertaken, but is usually too late to avail.

Sulphanilamide by mouth or by intravenous injections, especially in streptococcal infection.

Sulphapyridine (M & B 693) and sulphathiazole (M & B 760) are now beingused for pneumococcal and staphylococcal infections.

Concentrated scarlet fever antitoxin globulins—20 c.c. intramuscularly or intravenously—in severe cases is the best remedy in all streptococcal infections.

Intravenous, subcutaneous, or rectal injections of 6 to 8 pints of 10 per cent glucose solution daily help to eliminate the poisons.

Pyæmia.—Is caused by the diffusion of septic emboli throughout the circulation.

BACTERIOLOGY.—

STREPTOCOCCUS is the commonest cause. *Bacillus coli*, *B. pyocyaneus*, *Staphylococcus*, *Pneumococcus*, *B. typhosus* are all rare.

CAUSES.—Infective phlebitis—Osteomyelitis—Infective endocarditis—Middle-ear disease—Puerperal infections—Any septic focus. In all these there is a septic thrombus formed in the veins, particles of which become washed into the general circulation.

PATHOLOGY.—Thrombi containing living bacteria form emboli which are caught in the capillaries of different organs. The lungs suffer first, and then the kidneys, brain, liver, spleen, and large joints. In portal pyæmia the liver suffers first and most.

Infarctions occur as wedge-shaped hæmorrhagic areas at each point of infection.

Abscesses develop from each septic embolus.

Some degree of septicæmia usually coexists.

VARIETIES.—

IN ACUTE PYÆMIA there is also marked septicæmia, and death occurs before any of the abscesses have time to become prominent

IN CHRONIC PYÆMIA no bacteria can be found in the blood. The abscesses develop at intervals and attain a large size. They are often in bones or joints.

SYMPTOMS.—All those of septicæmia and sapræmia may be manifest.

RIGORS of extreme severity and regular recurrence are the distinguishing feature Dyspnœa, cyanosis, and sweating follow each rigor. They occur at intervals of one or two days.

AN INTERMITTENT FEVER, with rises to 104°–106°, accompanying the rigors, is also well marked.

LOCAL INFARCTIONS AND ABSCESSES appear about the end of the first week. The visceral infarctions are usually small, but may be notified by sudden stabbing pain or the development of pleurisy.

ABSCESSES, superficial or in joints, often develop rapidly and without any signs of inflammation. When opened there is no attempt at granulation or inflammatory repair.

DURATION.—Varies from ten days to several months.

POST-MORTEM SIGNS are those of septicæmia, together with: (1) Some primary focus, usually of a bony nature, in which necrosed bone is found with neighbouring veins filled by breaking-down septic clots; (2) The infarctions or abscesses in different organs

PROGNOSIS depends upon: (1) Accessibility of the primary lesion, (2) The early treatment of the primary focus; (3) The vitality of the patient and the virulence of infection; (4) The presence of septicæmia as indicated by bacteria free in the blood-stream.

TREATMENT.—

LOCAL.—Radical treatment of primary focus—Opening and scraping septic wounds—Scraping out septic bony cavities—Amputation of a limb—Opening and plugging a septic sinus. Ligature of the chief emissary vein (e g., internal jugular).

GENERAL.—Antistreptococcus serum—Tonic treatment by drugs and food.

TETANUS

A toxæmia due to a local infection by the *Bacillus tetani*.

Causes.—

PREDISPOSING.—Hot climates. Agricultural labourers and stable attendants are specially liable to contact with contaminated soil.

EXCITING.—A wound—generally septic, sometimes merely contused or punctured—is infected by the tetanus bacillus.

Bacillus.—Occurs in ordinary garden earth and in road sweepings. Consists of a delicate straight rod which develops a spore at one end (drum-stick bacillus) (*see Fig.* 1, p. 7). It is provided with flagella. It is a strict

Tetanus—Bacillus, *continued.*

anaerobe, but can grow in surface wounds where pyogenic cocci absorb all the oxygen. It stains by Gram's method—Grows in the depth of stab cultures—Its spores are very resistant to heat and chemicals—It produces no effect on inoculation unless the tissues are depressed by injury or toxic effect of sepsis.

Pathology.—The bacilli remain localized to the point of infection. The disease is a pure toxæmia produced by the absorption of toxins from the wound. The toxins travel to the central nervous system in the perineural sheaths. In the central nervous system the tetano-toxin acts something like strychnine as a powerful spasmodic poison.

POST MORTEM: Redness and congestion of the nerves leading from the infected focus. Softening and ecchymosis in the brain and cord. Molecular changes in the motor nerve cells. Rupture of muscles.

Symptoms of Acute Tetanus.—

INCUBATION PERIOD.—Three days to 15 days. Patients who have had prophylactic antitoxic serum may present an incubation period of 3 to 12 months. Any surgical operation on the wound area is liable to rouse up the latent infection.

EARLY SIGNS.—Stiffness and cramps in the jaw muscles, and in the neck.

SPASMS.—Trismus or lockjaw—Rigidity of the cervical muscles—Facial spasm (risus sardonicus)—Pharyngeal spasm causing dysphagia—Trunk muscles producing opisthotonus (backward arching), emprosthotonus (forward arching), or pleurosthotonus (lateral arching)—Abdominal muscles, especially the recti, are strongly affected—Respiratory muscles are attacked last.

CHARACTER OF SPASMS.—Strong, very painful, and continuous (tonic) contractions, lasting several minutes at a time. Both tonic and clonic, as opposed to strychnine poisoning, are determined by any sensory stimulus, e.g., light, sound, or a draught. Often cause rupture of the muscles.

TEMPERATURE is always high at the end (110°), and is usually raised throughout. Sweats are profuse.

CONSCIOUSNESS is unimpaired, and the expectation of the convulsions produces great mental suffering.

DEATH occurs in two to six days from exhaustion or asphyxia.

Chronic Tetanus is the milder form of disease. The incubation period is longer, fever is absent, and the convulsions are much more limited and less severe.

Cephalo-Tetanus is a special form following some head injuries. Facial paralysis occurs in conjunction with other symptoms, e.g., spasms. Pharyngeal spasm and mania are prominent, and give it a resemblance to hydrophobia.

Local Tetanus.—Seen in the muscles round a wound where the antitoxin has counteracted general infection but not enough to prevent local nerve involvement.

Diagnosis.—

STRYCHNINE POISONING causes similar symptoms, but of much more rapid onset. The muscles become quite relaxed between the spasms, and the hands are often affected.

HYDROPHOBIA.—The convulsions are clonic. Hallucinations and mania well marked. The muscles of deglutition and respiration are markedly affected.

SIMPLE TRISMUS, as from dental caries or affections of the temporo-maxillary joint, shows no affection of the neck muscles, and is generally unilateral.

Prognosis.—Is always bad, but when the incubation period is long there is a better chance of recovery. Whereas 96 per cent die when the incubation period is under ten days, only 55 per cent die if it is three weeks or over. Mortality has been greatly reduced by prophylactic inoculation.

Treatment.—

EXCISION of the infected wound should be employed as a prophylactic measure only. If tetanus develops, then the less the original wound is disturbed the better.

ANTITETANIC SERUM is a pure antitoxin, and has little effect on the toxins which have already combined with the cells of the central nervous system.

Antitoxic serum should be given:—

For prophylaxis.—1500 units subcutaneously: (*a*) At earliest possible moment in every case with an earth soiled wound; (*b*) Prior to any operation on a patient who has been exposed to infection within 12 months.

For cure.—200,000 units given intravenously, and if patient's condition is poor at the end of 7 days 50,000 units are given intravenously.

FEEDING is by a rubber tube behind the teeth or through the nose or rectum.

ABSOLUTE QUIET and avoidance of all sensory stimuli.

DRUGS.—Chloral, bromide, and chloroform. Chloretone in 10-gr. to 30-gr. doses, administered by the rectum. Chloroform may be needed if spasms are severe.

INTRASPINAL INJECTIONS OF MAGNESIUM SULPHATE.—One drachm of a 5 per cent solution injected into the spinal theca is of great value in stopping the convulsions. It is to be repeated daily or when the convulsions begin to recur.

ANTHRAX

The Bacillus Anthracis (*see Fig.* 1) is large and long: 5 to 20μ long and 1 to 1·5μ wide. It is aerobic, and liquefies gelatin. It forms spores when growing outside the body in the presence of oxygen. These spores are very resistant to chemicals and heat.

Infection usually takes place by the contact of butchers, graziers, leather or wool workers with the diseased sheep or cattle. In the latter it causes splenic fever, and the beasts, their carcases, and skin retain the infection. *Incubation Period*, 3 hours to 3 days.

Malignant Pustule is the result of cutaneous inoculation by anthrax. Usually occurs on the face, neck, or arms. (1) An angry red pimple; (2)

Anthrax—Malignant Pustule, *continued.*

A crop of vesicles upon an infiltrated base; (3) A central black slough surrounded by vesicles and a large zone of œdema, are the stages in its appearance, which it goes through in four or five days. Then the temperature rises and grave septicæmia is caused by the distribution of the bacilli. Special symptoms may be due to the involvement of the lungs, intestine, or nervous system.

Anthrax Œdema is a more virulent form of local infection, in which widespread œdema with multiple cutaneous sloughs precedes a rapidly fatal septicæmia.

Woolsorters' Disease is an anthrax septicæmia without external lesion. Usually the infection is by the lungs, and a severe PLEUROPNEUMONIA is set up. More rarely the intestine is the primary focus, and painful diarrhœa with passage of blood occurs, as the symptom of ENTERITIS. In either case the result is usually fatal.

Treatment.—Excision of local focus is only done if the serum cannot be obtained or if there has been delay in diagnosis and treatment. Early injection with SCLAVO'S SERUM.

Better to inject a protective barrier of serum round the lesion first of all, or MULFORD'S SERUM, 40–120 c.c. intravenously or intramuscularly.

NEO-SALVARSAN intravenously in doses of 0·6 g. daily, depending on the severity of infection.

HYDROPHOBIA

Cause.—Transmission of the virus by the bite or lick of a rabid animal, usually a dog.

Virus is said to be represented in one stage of development by so-called Negri bodies found particularly in the hippocampus major of the dog.

Rabies in the Dog occurs three to five weeks after infection. It manifests three stages: (1) An altered disposition, in which the dog sulks or is snappy; (2) A stage of maniacal excitement, in which it attacks any one it meets; (3) A paralysed condition affecting the hind limbs and jaw. The dog throughout has no dread of water, but drinks freely.

Hydrophobia in Man.—Incubation period is six weeks up to six months.

PREMONITORY STAGE.—A mental change—terror, delusions, and suspicions, with insomnia—is the first symptom group.

STAGE OF EXCITEMENT.—Clonic contractions of the tongue and pharynx, which later spread to other parts as general convulsions. Any sensory stimulus gives rise to these, especially the sight of water or any attempt to swallow.

Mouth is filled with tenacious mucus.

Respirations are irregular.

PARALYTIC STAGE.—Violent homicidal mania is a rare occurrence. Temperature, 100°–103°.

Death occurs in two to seven days from exhaustion or glottic spasm.

Post-mortem changes are: Inflammation of the medulla oblongata, with engorgement of the salivary glands.

Treatment.—The dog should be kept under observation until the diagnosis is certain.

The bitten part should be EXCISED or CAUTERIZED after a tourniquet has been placed on the limb above.

PASTEUR'S TREATMENT should be tried as soon as possible. It consists in injections of preparations from the spinal cords of artificially inoculated rabbits, beginning with those whose toxicity has been rendered weak by long desiccation, and later using more virulent preparations.

When the disease has manifested itself, sedatives—chloral, bromide, and chloroform—are the only palliation.

GLANDERS

Aetiology.—It is primarily a disease of horses and other allied animals, in whom it causes ulcerative lesions of the nasal mucous membrane. A specific organism, the *Bacillus mallei*, has been isolated, and can be grown outside the body on potato media. It is communicated to any who come in contact with the affected animals.

Symptoms in Man.—Incubation period 3–5 days. A pustular eruption breaking down into an ulcer occurs on the hands or face at the infected spot. The associated lymph-glands enlarge. The viscera and joints are infected, and the patient dies of acute septicæmia within about ten days.

A CHRONIC FORM may occur also. In this chronic abscesses affect the limbs, and suggest syphilis or tubercle.

Diagnosis is customarily made by the history of association with an infected horse. It may be assisted in doubtful cases by the injection of a sterilized culture of the *Bacillus mallei*, known as mallein. In case of glanders this is followed by a sharp febrile reaction.

Treatment.—Early and free excision of the local areas of infection.

CHAPTER V

GONORRHŒA.

Infection by Gonococcus.—

CHARACTERISTICS OF ORGANISM (*see Fig.* 1, p. 7).—A diplococcus, kidney-shaped—Stains easily—Gram-negative—Occurs in pus or epithelial cells—Grows with difficulty on blood-serum—Acids favour growth, Alkalis prevent it—It can penetrate intact mucous membrane—It prepares the way for ordinary pyogenic organisms.

SEAT OF INOCULATION.—Urethra (male or female)—Vagina—Rectum—Conjunctiva (especially in new-born).

Pathology.—The meatus and navicular fossa are lined by squamous epithelium, which is more resistant. Cocci invade the columnar epithelium of the penile urethra. A desquamation of the epithelium with leucocytic invasion results.

IN THE MORE SEVERE CASES the submucous tissue is invaded and becomes the seat of ulceration with deep infiltration by round cells. This forms a stricture later by its conversion into fibrous tissue.

IN THE MILDER CASES the epithelium is regenerated in about six weeks, the columnar epithelium being replaced by stratified.

THE URETHRAL GLANDS undergo similar changes or become the seat of cystic suppuration.

Onset.—Symptoms of gonorrhœal urethritis appear 2–8 days after infection.

SYMPTOMS.—Itching of meatus—Scalding micturition—Tenderness along course of urethra—Frequent micturition—Pain in perineum—Pain in back—Constipation—Some general malaise.

GLANDULAR ENLARGEMENT occurs in severe complicated cases, especially when ulceration exists. It usually affects the inguinal glands.

Varieties of Gonorrhœal Urethritis.—

1. ANTERIOR URETHRITIS (all cases begin as anterior urethritis).—In front of compressor urethræ muscle—Scalding micturition—First half of urine passed contains more shreds than second.

   COMPLICATIONS.—Bubo — Balanitis — Chordee — Cowperitis — Stricture (late stage).

2. POSTERIOR URETHRITIS.—(Occurs at any time)—Behind compressor urethræ—Hæmaturia—Frequency of micturition—Pain after micturition—Pain in perineum—Second half of urine contains more shreds than first half—Many casts of prostatic ducts are in urine. When posterior urethra is affected toxæmic manifestations are pronounced.

   COMPLICATIONS.—Cystitis—Prostatitis—Epididymitis—Vesiculitis.

3. GLEET.—Thin or gelatiniform discharge continuing for years after gonorrhœa.

   CAUSES.—(*a*) Posterior urethritis where organisms are harboured in prostatic ducts; (*b*) Stricture of urethra; (*c*) Granular patches, i.e., ulcers of urethra, gout, alcoholism, Cowperitis; (*d*) Repeated attacks.

Differential Diagnosis of Genital Gonorrhœa.—

IN MALE.—

Non-specific Urethritis.—Very rare—Organisms are Gram-positive. Usually clears up, but may be very resistant to treatment.

Foreign Body in urethra (by sound).

Urethral Chancre (by induration).

Prostatic Abscess discharging into the urethra.

IN FEMALE.—

Non-specific Vaginal Discharges are very common.

Leucorrhœa: Discharge comes from cervix uteri only.

Specific nature of discharge can only be determined by a demonstration of the gonococci.

Prophylaxis.—Only sure method is to avoid infection. Irrigations with 1 : 5000 potassium permanganate after intercourse. Recently prophylactic sulphapyridine.

Treatment of Gonorrhœal Urethritis in Acute Stage (frequent scalding micturition, free purulent discharge).—(*a*) General; (*b*) Local; (*c*) Drugs; (*d*) Vaccines; (*e*) Hyperthermy.

a. GENERAL.—

Rest. Copious fluid Diet, e.g., milk, barley-water.

Forbid meat, alcohol, tea, coffee, and sexual intercourse.

Drugs.—Alkaline diuretics and antispasmodics in a mixture such as:—

℞	Pot. Cit.	gr. xxx
	Pot. Bicarb.	gr. xv
	Tinct. Hyoscyam.	gr. xx
	Infus. Buchu	ad ℥ss

℥ss t.d.s. early in the acute stage.

b. LOCAL TREATMENT: IRRIGATION.—Still regarded by some as a useful part of the treatment in connexion with the use of chemotherapy. Irrigation of the anterior urethra is performed by Janet's method; the irrigator should not be raised more than 1½ ft. above the pelvis. Potassium permanganate of a strength 1 : 8000 is used as the irrigating solution. Irrigations given twice daily. Posterior irrigation is little used now except in those cases which are sulphonamide-resistant.

c. CHEMOTHERAPY.—The introduction of the sulphonamide group of drugs has been a great advance in the treatment of gonorrhœa. Sulphanilamide, sulphapyridine, sulphathiazole have been used with great success, usually in a dosage of 2 g. as the initial dose, followed by 4 g. daily for 4–5 days. Recently the less toxic sulphadiazine has been used in similar doses. Opinion is still divided as to the value of local irrigations of the urethra in conjunction with chemotherapy. If used, irrigations of potassium permanganate, 1 : 8000, are given twice daily for at least 10 days, even though the discharge has ceased.

d. PENICILLIN.—For those cases which are sulphonamide-resistant, penicillin gives good results. It may well be that penicillin will eventually replace all other forms of treatment. Intramuscular injections (15,000 units) into the buttocks are given at four-hourly intervals for 48 hours.

Treatment of Acute Gonorrhœal Urethritis, ***continued.***

e. VACCINES.—Use of vaccines in acute gonorrhœa is not recommended; a small proportion of cases show good results. There is the danger that the patient's natural immunity may be depressed temporarily in the so-called 'negative phase' resulting in severe complications.

f. HYPERTHERMY.—*See* p. 44.

Urethroscopy in Gonorrhœa.—

PRECAUTIONS.—Urethroscopy in gonorrhœa should not be used as a routine procedure, and never in acute cases. Its chief value is in the diagnosis and treatment of chronic conditions associated with gleet. A preliminary injection of 1 drachm of 5 per cent solution of novocain should be given, and the meatus must be slit when it is too narrow to allow the passage of a moderate-sized tube.

PATHOLOGICAL CONDITIONS OBSERVABLE.—

Dark red colour, with tendency to hæmorrhage.

Pale surface when the mucous membrane is sclerosed or absent.

Inflamed urethral glands, which may be cystic and appear as minute yellow dots.

Polypoid projections, which represent exuberant granulations or hypertrophied tags of mucous membrane.

Granular patches, which represent chronic or healing ulcers when the mucous membrane has been destroyed.

TREATMENT THROUGH THE URETHROSCOPE.—

Granular patches, polypi, and inflamed areas may be touched with solid silver nitrate or the actual cautery.

Attempts to divide a stricture will probably be inefficient if done through the urethroscope.

COMPLICATIONS

By Extension.—

FROM ANTERIOR URETHRA.—

SECONDARY INFECTION by pyogenic organisms, e.g., *B. coli* and *Staphylococci.*

BALANITIS AND BALANOPOSTHITIS.—If phimosis, slit foreskin.

TYSONITIS.—Rarely seen since the introduction of sulphonamides.

LACUNAR ABSCESS.—Penile fistula. Open from outside as early as possible.

CHORDEE.—Painful erections, usually at night. Penis is curved with the concavity downwards because the corpus spongiosum does not dilate proportionately to the corpora cavernosa. Treat by bromides and cold compresses.

WARTS on genitals, especially in women.—Touch with nitric acid, cauterize, or dust with calomel.

INFLAMMATION OF THE PARA-URETHRAL DUCTS.

COWPERITIS.—Inflammation and suppuration of Cowper's glands, situated between the layers of the triangular ligament in the perineum. Usually one-sided. An abscess forms, which bursts externally by the side of the bulb, or rarely into the rectum. Treatment by fomentations and incision.

RETENTION OF URINE (spasmodic and congestive).—Treat by hip-baths, sedatives, and, if necessary, soft catheter.

STRICTURE.—Generally a late sequela.

GRANULAR ULCERS.

FROM POSTERIOR URETHRA.—

IN THE MALE.—

Epididymitis (*see* Chap. XLVII) usually occurs in the second or third week, or in the chronic stage. Urethral discharge ceases or diminishes on its occurrence. It may be caused by injudicious injections in the acute stage. Usually one-sided, but both sides may suffer successively. Bilateral affection generally leads to sterility.

Treatment: Suspensory bandage, with lead and spirit lotion. In severe cases, rest in bed, with hot fomentations. Discontinue any local urethral treatment. Continue treatment with sulphonamide group of drugs.

Cystitis and Prostatitis probably occur in all cases in a limited degree, certainly in all cases of posterior urethritis. Rarely these conditions are acute and suppurative, leading to prostatic abscess or diffuse urinary infection. These are generally cases of secondary infection, and occur in those who have an old stricture.

Vesiculitis, or inflammation of the vesiculæ seminales, probably occurs in most cases in conjunction with prostatitis and epididymitis. It may suppurate and cause a perineal or pelvic abscess. Rarely it causes blood-stained spermatic emissions.

IN THE FEMALE.—Cystitis—Labial abscess—Vaginitis—Endometritis—Salpingitis—Ovaritis—Peritonitis, generally localized in pelvis, and causing adhesions and sterility, sometimes general and acute.

IN CHILDREN.—In female infants vulvo-vaginitis occurs which may lead to peritonitis or arthritis and pyæmia. In some cases general infection begins with a gonorrhœal stomatitis.

By Transmission of Virus.—

PROCTITIS.—Tenesmus and discharge. Treat by irrigations and astringents.

RHINITIS.—Treat by alkaline irrigations.

CONJUNCTIVITIS.—

In adult (unilateral).—Treat by shield over good eye—Irrigate with boracic acid—Instil argenti nitras, gr. v ad ℥j, quartis horis—Cover with iced compresses.

In infants (ophthalmia neonatorum—bilateral). Often causes corneal ulcers, corneal opacities, perforation of the eye, blindness. Treat as above, cutting the external tarsal commissure if necessary for drainage.

By Absorption.—

General infection may arise in three ways: (1) By the absorption of the gonotoxin; (2) By the gonococcus; (3) By the pyogenic organisms of mixed infection—*staphylococci*, *streptococci*, or *Bacillus coli*.

General infections are commonest in males as a complication of posterior urethritis. They occur late in the disease or in the second or third week of the acute stage.

GONORRHŒAL RHEUMATISM occurs in three forms:—

1. ARTHRALGIA without inflammation, but with myalgia and tenosynovitis.
2. HYDRARTHROSIS.—Painful effusions into the joints, especially the knees, associated with inflammation of peri-articular fibrous structures, e.g., tendon sheaths, ligaments, and bursæ.

3. **True Arthritis** of a sero-fibrinous or purulent type, the former being the most common. The onset is usually sub-acute and polyarticular, attacking the knees, ankles, and wrists most often. Often all affected joints recover quickly except one, which remains inflamed.

Fibrous ankylosis is a common result and bony ankylosis a rare one.

Diagnosis.—

In Acute Rheumatism.—There are many joints affected, migrating from one to another. The pain is acute, even in the absence of movement. Fever and sour sweats are present. Pain relieved by salicylates.

In Tuberculous Joints.—The onset is more gradual and there is less inflammation. It is limited to one joint.

AFFECTIONS OF MUSCLE, NERVE, AND FIBROUS TISSUE.—With or independently of arthritis, various characteristic painful affections may occur.

Pain in the Heel and Sole of the Foot from affection of the plantar fascia, ligaments, and tendons.

Tenosynovitis and Bursitis, chiefly of the tendons of the ankle and wrist.

Lumbago, Sciatica, Neuritis.

GENERAL BLOOD INFECTIONS.—

Septicæmia and Pyæmia are very rare, and are then probably due to mixed infection.

Endocarditis and Pleurisy are rare results of a gonococcal septicæmia.

KERATODERMIA BLENORRHAGICA (GONOCOCCAL HYPERKERATOSIS).—May occur a long time after the original urethritis. Affects the soles of the feet; lesion is a papulo-pustular one, and the lesions become covered with horn-like crusts, resembling closely the rupial lesions of syphilis. Usually has an associated arthritis; conjunctivitis, iritis, and endocarditis occasionally accompany the condition. Patients usually very ill. Hyperpyrexia, using the Kettering hypertherm, clears up skin lesions and arthritis. Elastoplast to the affected areas is very useful.

TREATMENT of general infections.—

In the Milder Cases which are probably due to an absorption of toxins, the local condition should be energetically treated by injections, or through the urethroscope and by chemotherapy.

In the Severe and Chronic Cases:—

Artificially induced pyrexia followed by a course of chemotherapy has been found of great value in treating chronic cases.

Pyrexia induced either by:—

1. Intravenous T.A.B. vaccine commencing with dosage of 25×10^6 bacteria increasing to 100×10^6 bacteria, depending on the reaction and the patient's condition.
2. Kettering hypertherm. Patient kept at a temperature of 106° F. for 5 hours in a specially insulated cabinet. For details see standard works on the subject.

CHAPTER VI

SYPHILIS

Definition.—An infective venereal disease peculiar to human subjects but inoculable into animals.

Cause.—Infection by a specific spirochæte, the *Treponema pallidum*. Inoculation of any thin or abraded skin or mucous membrane.

Pathology.—

BACTERIOLOGY.—THE TREPONEMA PALLIDUM is now regarded as pathogenic. It is 4 to 20μ in length and about $\frac{1}{4}\mu$ wide, with flagella at either pole. The turns in the spiral are set very close, and number from 8 to 12. It is motile. Noguchi and others have cultivated the organism in the test-tube in pure state. It is best demonstrated in the primary sore by dark-ground illumination or by Burri's Indian-ink method.

DISTRIBUTION.—In all primary and secondary lesions and on the ulcerated surfaces. Lymph-glands, saliva, and urine. Ovary, placenta. Very numerous in stillborn fœtus of syphilitic mothers. It is very scarce in gummata.

IMMUNITY.—Animals enjoy a NATURAL IMMUNITY against syphilis except in the case of the higher apes. Races in which syphilis has been common for many generations have acquired probably a RELATIVE immunity compared with virgin races, in which it assumes a very malignant form.

ACQUIRED IMMUNITY of a limited kind is conferred by an attack of syphilis. Within ten days of the appearance of the chancre other chancres may be produced by auto-inoculation. A second attack of syphilis may be acquired a few years after the first, showing that acquired immunity is not lasting. Patients with hereditary syphilis may acquire the disease after puberty.

WASSERMANN'S REACTION.—This is based on the supposition that the syphilitic toxin (antigen) caused the formation of an immune body of the order of 'amboceptors' that required the 'complement' to satisfy it. By mixing antigen (extract of syphilitic organs), amboceptor (syphilitic patient's serum), and complement (normal animal serum), the *complement* is fixed and cannot act with another antigen-amboceptor mixture, i.e., red blood-cells + anti-red-cell (hæmolytic serum from an animal). The second or hæmolytic mixture is not satisfied in the absence of free complement and cannot lake red cells. Where the complement is not fixed by the first mixture, hæmolysis will occur. Thus the absence of hæmolysis indicates syphilis.

It has since been discovered that the reaction probably depends upon a perverted metabolism leading to the appearance of certain colloidal bodies in the blood, and that any tissue will provide the antigen. Nevertheless there is still a considerable degree of specificity in the reaction.

Syphilis—Pathology, *continued.*

TISSUE CHANGES.—In all cases there is a marked connective-tissue reaction, especially round the vessels.

In Chancre.—Large accumulation of lymphocytes and plasma cells. Some lymphangitis and endarteritis.

Secondary Lesions.—Begin round the blood-vessels. There is a marked proliferation of the cells of the interpapillary processes of the epidermis.

Gummata.—A large mass of connective tissue and plasma cells are enclosed in a fibrous capsule—They undergo fibrosis or necrosis—There are some giant cells—The vessels show marked peri- and endarteritis, which probably accounts for the tendency to 'gummy' degeneration.

Sources of Infection.—

Primary Lesions.

Secondary Lesions, especially condylomata.

Indirectly, e.g., by pipes or drinking vessels used by those with lesions of mouth.

Stages of Syphilis.—

PRIMARY.—Local manifestation at the seat of inoculation. Absent in inherited syphilis

SECONDARY—General infection. Symptoms occur from one month to two years after inoculation. Lesions are superficial, symmetrical, and infective.

TERTIARY.—Lesions appear from second year onward. They consist of deep fibrocellular infiltrations of skin, bones, and viscera. They are not infective.

Incubation Period.—9 to 90 days.

Primary Lesion.—Hard or Hunterian chancre.

POSITION in order of frequency.

Penis.—Glans or inner surface of prepuce—Outside skin—Meatus (common)—Urethra (very rare).

Vulva, vagina, or cervix.

Lips, anus, fingers, tongue, breast, abdomen, palate, or tonsils.

CHARACTERS OF HARD CHANCRE.—Usually single—Appears 2 to 6 weeks after infection—Begins as a papule, which ulcerates later—Base is indurated like thin cartilage—Painless and without inflammation—Associated lymph-glands become 'shotty', i.e., enlarged, but remaining painless and discrete and freely movable—Lymph-vessels between chancre and glands are often thickened and indurated.

It is followed by signs of specific syphilitic infection. Always make a microscopic examination of the secretion. Presence of *Treponema pallidum* is a proof of the nature of a hard chancre.

Heals spontaneously.

Leaves little or no scar.

DIAGNOSIS OF PRIMARY SYPHILIS.—

Soft Chancre.—Generally multiple—Appears 3–6 days after infection—Consists of a deep ulcer, which rapidly extends, and has sloughy base—Copious foul discharge—All signs of inflammation—Lymph-glands become inflamed and matted together and suppurate—No induration except inflammatory exudation—Will not heal without local treatment—Leaves well-marked scar—Is not followed by general infection.

Chancre of Mixed Infection.—Presents signs of soft chancre at first—Later becomes indurated, and is followed by signs of secondary syphilis.

Primary Syphilis and Gonorrhœa.—Inflammatory complications of gonorrhœa may mask hard chancre—Indurated sore, followed by secondary syphilis.

Concealed Chancre.—Phimosis may conceal sore and produce a good deal of discharge—Bullety bubo and later secondaries.

Epithelioma.—Patient generally over 50—Longer history—Foul discharge—Warty growth with hard base, or ulcer with everted hard edges.

Gumma or 'Relapsing Chancre'.—Associated with other tertiary lesions—Past history of primary and secondary syphilis—Inguinal glands not enlarged.

Psoriasis—Boil—Inflamed sebaceous glands—Eczema—Lupus.

COMPLICATION OF CHANCRE.—Phagedenic ulceration, especially when occurring under a tight prepuce.

TREATMENT OF PRIMARY SYPHILIS.—

No local treatment until *Treponema pallidum* demonstrated in chancre.

If chancre is **Dry**: Rub with calomel ointment (30 per cent).

If chancre is **Moist, Ulcerated,** or **Soft**:—

Lotio nigra or iodoform ʒj, boracic acid ad ℥j.

If chancre is **Foul** and **Rapidly Spreading**:

Acid nitrate of mercury.

If chancre is seen within a few days of its appearance, and is situated **on the Prepuce**:

Excise by circumcision.

If chancre is concealed by **Phimosis**:

Slit open prepuce.

Rigorous constitutional treatment as soon as diagnosis is certain.

Secondary Syphilis.—

APPEARS one to two months after chancre, two to three months after infection.

LASTS any time up to two years from infection.

GENERAL CHARACTERS OF SECONDARY LESIONS.—Lesions affect skin, mucous membranes, lymph-glands, and eyes—Copper colour—Polymorphic—Roughly symmetrical—Tend to spontaneous recovery—Without pain or irritation (secondary ulcers and fissures in mouth are painful).

GENERAL SYMPTOMS, which appear just before eruptions.—

Anæmia.

Aching Pains in bones and loins, worse at night.

General Enlargement of the Lymphatic Glands.—Most noticeable in posterior triangle of the neck and internal epitrochlear region.

A **Febrile Attack**, occasionally lasting a few days.

Cephalagia (*see under* **Tertiary Lesions**).

SKIN ERUPTIONS OF SECONDARY SYPHILIS.—

1. **Roseola or Erythema.**—

Chiefly over trunk and flexor surfaces of limbs.

Patchy erythema fading on pressure.

Often leaves a patchy pigmentation, especially round the neck of women.

Secondary Syphilis—Skin Eruptions, *continued.*

2. **Papular.**—

All over the body. Very characteristic round forehead: 'corona veneris'.

a. Squamous or Psoriasis: When papules are covered by scales of desquamating epithelium.

Squamous syphilides are very characteristic on the palms and soles. The scaly skin becomes heaped up and fissured.

b. Condylomata, or moist papules on the skin: Papules present on surfaces of skin which are kept habitually moist. Occur in order of frequency: Round anus—Scrotum—Perineum—Vulva—Lips—Umbilicus—Axillæ—Under breasts—Between toes—External ear.

These erythematous and papular eruptions are the only ones seen at all commonly. They are quite superficial, and leave no scar.

3. **Pustules.**—Resembling acne—When a number of pustules are close together, and break, and become covered by a large scab, it is termed ecthyma.

4. **Bullous.**—Pemphigus—Large vesicles which suppurate, break, and are covered by a conical crust.

5. **Rupia.**—Deep ulcers covered by limpet-shaped scabs.

Ecthyma, pemphigus, and rupia only occur late in very severe cases. Pemphigus is very characteristic of a severe form of congenital syphilis. They all leave scars which are characterized by being circular or gyrate in outline, thin, supple, and white.

6. **Pigmentary Syphilides.**—Syphilitic leucoderma seen in the secondary stage and affects the neck and shoulders. Lesion is hyperpigmentation with areas of depigmentation. May be due to syphilitic damage to suprarenal cortex.

AFFECTIONS OF THE HAIR AND NAILS.—

Alopecia.—

1. General shedding of the hair and eyebrows during secondary stage—Yields to general treatment.
2. Patches of hair follicles are destroyed by deep ulceration, either secondary, tubercular, or tertiary—Incurable.

Onychia.—Nails may become grooved, brittle, and broken in late secondary syphilis.

Perionychia.—Secondary ulceration round nail margin—May cause nails to be shed.

AFFECTIONS OF THE MUCOUS MEMBRANES OF THE MOUTH AND THROAT.—

Simple Congestion of faucial mucous membrane.

Bald Areas on tongue.

Mucous Patches, i.e., papules on mucous surface.

Fissures, especially near angle of lips and edges of tongue.

Ulcers.—Superficial, covered by grey membrane on tonsils and fauces. They spread by narrow grey margin—the 'snail-track ulcers'.

Œdema Glottidis occasionally complicates ulcerations at the back of the mouth and pharynx.

Stomatitis due to Mercury.—Spongy bleeding gums—Profuse salivation—Fœtid breath—Ulcers with dark sloughy bases.

Treatment of Oral Secondary Syphilis.—

Avoid alcohol and tobacco.
Cleanse teeth carefully.
Gargles of alum or chlorate of potash.
Touch ulcers with chromic acid, gr. x ad ℥j.

SECONDARY AFFECTIONS OF THE EYES.—

Iritis: Marked congestion, effusion, and adhesions—Pain slight.
Choroiditis, Retinitis (both rare).

LATE SECONDARY LESIONS—sometimes termed 'reminders'—sometimes forming intermediate or early tertiary signs.—

Peeling patches on palms and soles—'syphilitic psoriasis'.
Symmetrical painless epididymitis.
Synovitis, especially of knees.
Osteocopic pains, or periosteal nodes, often in evidence.

Tertiary Syphilis.—

Lesions occur generally from second year after infection onward to an indefinite period.
Are non-infective.
Do not cause secondary lymph-gland enlargement.
Asymmetrical.
Affect the skin and deep tissues, especially Fibrous tissues—Bones—Viscera.
Tend to (*a*) Deep ulceration; or (*b*) Formation of a gumma; or (*c*) Fibrosis, i.e., hypertrophy of connective tissue and atrophy of parenchymatous tissue of affected organs.
No tendency to spontaneous cure.
Are generally amenable to iodides.

CHARACTERS OF A GUMMA.—Begins as collection of small round cells around blood-vessels in connective tissue. Blood-vessels are occluded by: (*a*) Endarteritis obliterans; (*b*) Pressure of small-celled infiltration. Central mass of cells dies and forms central 'gummy' core. Suppuration generally supervenes in superficial gummata. When skin or mucous membrane has burst, a 'wet wash-leather slough' is seen. Healing involves deep scarring with great contraction. Is generally painless and without signs of inflammation.

GUMMATOUS ULCER.—Formed by bursting of superficial gumma. History of preceding lump. Deeply punched out edges. Surrounding tissues healthy. Base formed by 'wash-leather' slough. Granulations are firm, fibrous, and avascular. Outline is often formed by coalescing circles formed by fusion of small gummata. Painless. Scars left are thin and supple, with pale centre and pigmented margin.

TERTIARY LESIONS OF THE SKIN.—

Gummata, probably of subcutaneous origin—Commonest in region of knees—Loins—Sacrum and buttocks.

Lupoid Ulceration.—Tuberculous masses in the skin coalesce, ulcerate at one part and heal at another—Serpiginous outline—Especially attacks face, but may occur anywhere—Copper-red colour when active—Supple white scar when healed.

TERTIARY LESIONS OF THE BONES.—

Periostitis.—Local, plastic, forming 'hard nodes'—Diffuse, combined with sclerosing osteitis, causing osteosclerosis (*Fig.* 16).

Gummata.—Subperiosteal, forming 'soft nodes'—Central, causing expansion with possible spontaneous fracture.

Tertiary Syphilis—Lesions of Bones, ***continued.***

Necrosis or Caries.—Probably resulting (*a*) from blood-supply being cut off by gummata or osteosclerosis, or (*b*) by secondary septic processes.

Special Bones Affected.—

Nasal.—Necrosis, perforation of septum, sunken bridge.

Hard Palate.—Perforation into the nasal cavities.

Cranial Vault.—Especially frontal and parietal bones—One or more circular ulcers, or extensive 'worm-eaten' surface, black colour, necrosing fragments—Great thickening from osteosclerosis.

Tibiæ, clavicles, femora, forearm bones.

Sternum.

Phalanges.—Especially proximal, causing syphilitic dactylitis—A local, hard, periosteal thickening occurs in shaft of bone—It causes shortening of the bone, but seldom involves the joint—Painless, and rapidly heals under iodides.

To be diagnosed from tuberculous dactylitis, which begins as central swelling at epiphysial end of bone—Is very painful—Involves joints—Is not influenced by iodides.

LIPS, TONGUE, AND PHARYNX.—

Leucoplakia.

Fissures producing deep scarring and contraction.

Gummata.—Commonest in dorsum of tongue—May destroy soft palate or produce extensive adhesions or pharyngeal stenosis.

LARYNX.—

Hyperplasia of epiglottis or aryteno-epiglottidean folds.

Gummata and gummatous ulceration.

Perichondritis and Necrosis of epiglottis or arytenoids, or rarely cricoid and thyroid cartilages.

Sometimes complicated by œdema glottidis.

RECTUM.—

Gummata surrounding bowel, ulcerating into its cavity—Forming fistulæ—Producing commonest form of non-malignant stricture.

CARDIOVASCULAR SYSTEM.—The valves of the heart may be affected, causing valvular disease. The large arteries are affected by obliteration of the vasa vasorum, causing a fibrotic degeneration of the tunica media, with loss of elasticity. This may lead to *aneurysm* of the aorta or any of the large arteries.

VISCERA.—In order of frequency: Testis, liver, spleen, heart, lungs, and any of the other viscera rarely.

Two forms: Softening Gummata and Fibrous Thickening.

NERVOUS SYSTEM.—

Cephalalgia, which may be marked in the late secondary or in the tertiary period, is characterized by intensity, persistence, and liability to nocturnal exacerbations.

Brain.—Gummata (usually begin in bones or meninges)—Obliteration of cerebral arteries—Aneurysm of cerebral arteries—Chronic meningitis—Paralysis of nerves, especially second, third, fourth, sixth—General paralysis of the insane.

Spinal Cord.—Chronic meningitis—Subdural gummata—Locomotor ataxy.

Charcot's Joints or perforating ulcer of the foot commonly result from syphilitic tabes.

Congenital or Hereditary Syphilis.—

MODES OF ORIGIN.—

PATERNAL.—Infection accompanying the spermatozoon. About 40 per cent of children thus infected are stillborn or die in early infancy.

MATERNAL.—The mother has syphilis before conception. The ovum is primarily infected. The child mortality is about 80 per cent.

MIXED INFECTION.—Both parents have syphilis. The child mortality is about 90 per cent.

MANIFESTATIONS.—Congenital syphilis may manifest itself in one of the following ways:—

1. Miscarriage during the early months of pregnancy.
2. Birth of a stillborn macerated fœtus.
3. Child presents obvious syphilitic features—wasting, snuffles, etc.
4. Child is apparently healthy at birth, but develops syphilitic stigmata during the first few weeks of life.

COLLES'S LAW.—A healthy mother who begets a syphilitic child cannot be infected by the latter.

PROFETA'S LAW.—A healthy child born of a syphilitic mother cannot be infected during infancy. This immunity does not apply to the acquisition of syphilis in adult life.

FŒTAL SYPHILIS.—Usually produces abortion and stillbirth—The fœtus may be macerated—The placenta, liver, lungs, heart, and vessels are the seat of small-cell infiltration and are full of spirochætes.

INFANTILE SYPHILIS.—The child is syphilitic at birth—Marasmus and cachexia make it wizened and monkey-like—The spleen and liver are enlarged—Hydrocephalus or microcephaly may be present.

SYMPTOMS of congenital syphilis appear generally within three months of birth—Are those of secondary and tertiary disease, with following special features:—

EARLY RASH specially affects buttocks and genitals.

ULCERS round mouth, nares, and eyes leave radiating scars.

PEMPHIGUS is specially characteristic of a grave form It may affect even the palms and soles.

GENERAL STOMATITIS, producing early loss of milk teeth and deformity of permanent.

OTITIS MEDIA, with permanent deafness

ORCHITIS, producing a hard swelling of the testes on both sides, with atrophy later. It occurs within six months of birth; this and its bilateral character distinguish it from tuberculous orchitis of children.

PURULENT RHINITIS, producing 'snuffles', loss of bridge to nose in later life (*Fig.* 17), caries of spongy bones, with ozæna.

HIGH PALATE (due to disease or stunted growth of nasal septum).

CRANIUM.—Craniotabes: Uncommon to get this. Unossified spots in frontal or parietal bones. 'Parrot's nodes' over the frontal and parietal eminences, causing natiform or 'hot-cross-bun' skull.

Both these lesions may result from, or be intensified by, severe rickets.

LONG BONES, especially humerus, tibia, radius, and ulna.—Diffuse periostitis seen in the long bones after the child has begun to walk; most typical is the so-called *sabre-shaped tibiæ*.

Epiphysitis, producing thickening—Separation of epiphysis—'Pseudo-paralysis'—Suppuration in joints, the acute arthritis of infants.

Congenital Syphilis—Symptoms—Long Bones, ***continued.***

There is an irregular proliferation and fatty degeneration of the cartilage cells, with defective ossification of the growing end of the bone.

JOINTS may be affected.—(1) Chronic synovitis; (2) Symmetrical osteoarthritis; or (3) Suppuration secondary to epiphysitis.

LATE SYMPTOMS, occurring especially during puberty and adolescence:—

GUMMATOUS and LUPOID ulceration, most usually round nose, mouth, or knees.

INTERSTITIAL KERATITIS occurs usually from 5 to 20—Both eyes affected, but one at a time—Cornea is infiltrated with round cells—Ground-glass opacity—Marked circumcorneal zone of congestion—Vascular patches on cornea ('salmon patches')—Tends to recover—Leaves white patches.

DEAFNESS due to labyrinthine disease.

SCLEROSING OSTEOPERIOSTITIS or epiphysitis of long bones. May produce massive thickening, lengthening, or shortening. These lesions are often symmetrical.

AFFECTIONS OF TEETH (*Fig.* 18).—In permanent set in order of frequency: Central upper incisors, lateral upper incisors, lower incisors show:—

Cutting edge or crown smaller than base, forming 'peg-top' teeth.

Semilunar notch in cutting edge (*Hutchinson's teeth*).

Upper central incisors are widely separated.

The first permanent molars are ill developed and dome-shaped, forming the so-called *Moon's teeth.*

PARASYPHILITIC PHENOMENA.—Idiocy, meningitis, encephalitis, spastic paraplegia, hydrocephalus.

Prophylaxis of Syphilis.—

An inunction with calomel ointment (10 parts calomel, 20 parts lanolin) will prevent chancre formation if used within two hours of exposure to infection.

EXCISION OF A CHANCRE has no influence in preventing the development of the disease.

Treatment of Syphilis.—Treatment of syphilis falls into three groups: (1) General. (2) Local. (3) Specific.

1. GENERAL.—Hygienic surroundings—adequate food—tonics. Iron in the secondary stage to combat anæmia. Attention to oral and dental hygiene when receiving bismuth or mercurial preparations.

2. LOCAL.—

Chancre—nil until dark ground reveals *Treponema pallidum.* Later, treat with weak antiseptic washes and apply 15 per cent calomel ointment.

Mouth-washes in oral manifestations containing mercurial derivatives.

Condylomata—general cleanliness and dusting powders: calomel, starch, and boracic.

Gummatous ulcers.—Keep clean and bathe with mercurial lotions.

3. SPECIFIC.—

ARSENIC.—Various preparations have been used, neosalvarsan (N.A.B.), and, recently, less toxic mapharside. Give intravenously, and use freshly prepared solutions. Patient examined carefully, especially with reference to cardiovascular system. Urine examined for bile and albumin. No food for three hours before injection. Purgative the night before injection. Various standard courses in use. Wise to start with small doses, as 0·9 g. mapharside, two injections in first week, then 0·06 g. weekly for a course of ten injections. Followed by a month of bismuth and iodides, and then further courses of ten injections alternating with bismuth for at least one year. Recently intensive arsenical therapy has been used, patient receiving up to 1300 mg. in ten days. Not without danger. Toxic effects of arsenic are:—

1. Extravenous injection—local ulceration and sloughing of tissues.
2. Herxheimer Reaction.—'Therapeutic shock' following liberation of syphilitic toxin after first injection of arsenic. Rigors, fever, malaise, severe reactions if myocardium and aorta are at all involved in the syphilitic processes. May prove fatal.
3. Nitritoid crises.
4. Dermatitis.
5. Jaundice.
6. Hæmorrhagic encephalitis.

BISMUTH.—Given as intramuscular injections of the hydroxide (0·2 to 0·3 g.). It may produce albuminuria or stomatitis.

MERCURY.—Whether by mouth (calomel) or by inunction, vaporization, or intra-muscular injections, mercury has now been superseded by the preparations of arsenic and bismuth.

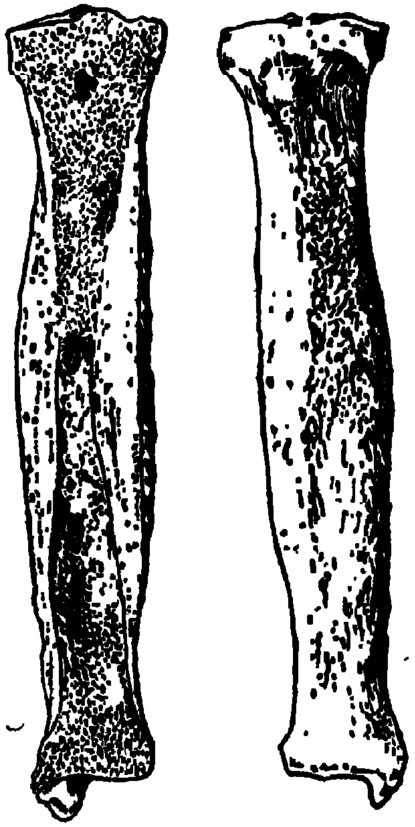

Fig. 16.—Syphilitic osteosclerosis of tibia. The whole bone is thickened and heavy, the marrow cavity being encroached on by increase of the cortex. A late tertiary manifestation

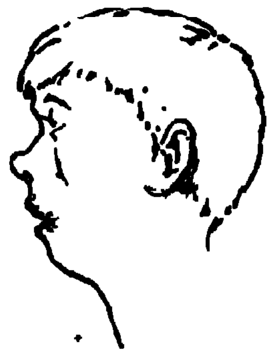

Fig. 17.—Facies of congenital syphilis. Saddle-shaped bridge of nose. Low forehead. Scars at the angle of the mouth.

Fig. 18.—Teeth in congenital syphilis (Hutchinson's teeth).

Treatment of Syphilis, ***continued.***

IODIDES.—Only necessary when tertiary symptoms are present.

Act by causing small-cell infiltration to be absorbed.

Dose, gr. v, increased to gr. xxx t.d.s., or until action begins to be apparent. Give with ammonium carbonate gr. v, and plenty of water.

IODISM, or the irritative and toxic effects of iodides, consists in coryza and an acneform eruption. Coryza is more likely to occur after small doses than after large.

Iodides should generally be given only for three or four weeks at a time.

GENERAL PARALYSIS OF THE INSANE.—Treated by malarial inoculation and pentavalent arsenic—e.g., tryparsamide.

For treatment of cardiovascular syphilis and syphilis of nervous system, see standard text-books of medicine.

Safety for Marriage.—Is only safe four years after infection if treatment has been efficient, if no symptoms have been present for two years, and if the Wassermann reaction has been repeatedly negative.

CHAPTER VII

TUBERCULOSIS AND ACTINOMYCOSIS.

TUBERCULOSIS

Aetiology.—

PREDISPOSING CAUSES.—

INHERITED SUSCEPTIBILITY, especially flat or narrow chests—'Sanguine and phlegmatic dispositions'.

CHILDREN and young adults are specially susceptible.

ATMOSPHERIC CONDITIONS.—Bad ventilation, overcrowding, and dusty occupations are very potent causes.

ANY LOCAL WEAKENING of the tissue resistance.—Chronic catarrhal conditions—Strains, sprains, especially of bones and joints.

THE EXCITING CAUSE is the infection by the tubercle bacillus.

Tubercle Bacillus.—4–5μ long and 0·2–0·3μ wide. Grows on blood serum, glycerinated agar-agar, or Lowenstein's medium. Grows very slowly, first as small dots, then as wrinkled layer. Filamentous forms, often branched, occur. Stains with difficulty, but retains the stain against extraction with acids, i.e., is acid-fast. (*See Fig.* 3, p. 7.)

THREE TYPES.—

1. AVIAN, with no relation to human disease.
2. BOVINE.—Conveyed by infected milk, causing lymphatic, peritoneal, bone, and joint disease. The most usual type in children.
3. HUMAN.—Conveyed by air, causing pulmonary and miliary infection. The usual type in adults.

Methods and Channels of Infection.—Through respiratory organs by breathing tubercle-laden dust. Through alimentary organs by eating tubercle-infected food. Through injuries and abrasions of the skin or mucous membranes. Through lymph-channels, i.e., tonsil, bowel.

Histology of a Tubercle (*Fig.* 19).—The grey miliary tubercle is smallest tubercle visible to naked eye. It consists of a number of submiliary tubercles or giant-celled systems. Each giant-cell system consists of:—

CENTRALLY-PLACED GIANT CELL.—Measures 50–500 μ. Many (20–50) nuclei massed near periphery. Bacilli are in the cell opposite to nuclei. Protoplasm is always degenerate: coagulation necrosis or caseation. Not to be confused with macrophages and the giant cells of the bone-marrow.

EPITHELIOID CELLS.—Placed in a zone round giant cells. Each is two to three times as large as a white cell. Elongated, with oval nucleus. Contain tubercle bacilli in or between them. Most characteristic feature of tubercle.

LEUCOCYTES.—Form a peripheral zone. Contain no tubercle bacilli. Result from inflammatory reaction round tubercle.

RETICULUM.—Fibrillar network between cellular elements.

Tuberculosis, *continued.*

Later Stages in the tuberculous process.—

CASEATION.—A number of miliary tubercles join to form a mass formed of all three kinds of cells. Blood-vessels being scanty or absent, necrosis takes place. This cell necrosis produces a cheesy structureless mass, known as caseous material. Caseation is best marked near the centre of the tubercle.

CICATRIZATION.—This is a process of repair. The granulation tissue surrounding the caseous centre is converted into fibrous tissue, and contracts. The liquid parts of the caseous material are absorbed, and the remainder forms a chalky mass—the so-called calcareous transformation.

LIQUEFACTION OR SUPPURATION.—This is similar to ordinary suppuration, but it takes place without the usual signs of inflammation. A chronic abscess is formed, i.e., cold abscess. If this lesion is near the surface it may burst, the contents of the abscess are discharged, and a tuberculous ulcer is formed. A lesion which is more deep may track along fascial planes and then become superficial and burst, leaving a tuberculous sinus.

PYOGENIC SUPPURATION.—In localities, e.g., the lung, where free access to the air occurs, or in any lesion where contamination from the skin or alimentary tract has taken place, a true pyogenic infection is set up which rapidly destroys the tissues and produces all the signs of pyogenic absorption.

Spread of Tuberculosis.—

BY LOCAL EXTENSION.—The disease spreads by direct contiguity from one organ to another, e.g., from the lung to the pleura or from a bone to the joint.

BY METASTATIC DEPOSITS.—Conveyed by blood, lymph, or secretions. Pulmonary tubercle often arises from that of the joints, tubercle of the bladder from that of the kidney, tuberculous meningitis from tubercle of the testis.

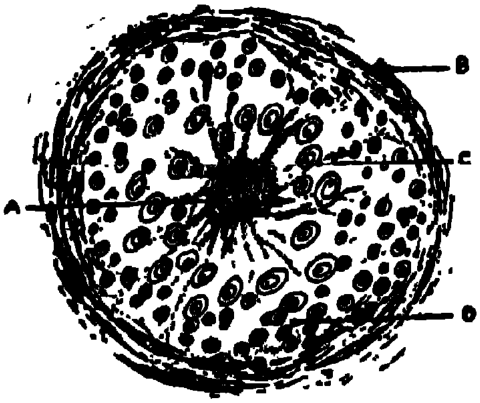

Fig. 19.—An early tubercle before caseation (diagrammatic). A, Central giant cell with peripheral nuclei; B, Zone of fibroblasts; C, Zone of endothelioid cells; D Zone of lymphocytes.

BY GENERAL BLOOD DISSEMINATION.—**A general miliary tuberculosis arises from the development of tubercles round all the small vessels, especially in the serous membranes.**

Treatment.—As a general principle, this is a disease from which recovery will take place naturally, provided that the proper constitutional and local conditions be observed.

CONSTITUTIONAL CONDITIONS NECESSARY FOR RECOVERY.—

Fresh Air, especially dry, bracing, open air. Sunlight, or Ultra-violet Light Therapy. Administration of Vitamins. Abundant Nitrogenous and Fat Food.

LOCAL CONDITIONS NECESSARY FOR RECOVERY.—

Rest for the diseased part, both physical and functional. Hence the difficulty in treating tubercle in parts like the lung or bladder, which can never have complete physical or functional rest.

Absence of Pyogenic Infection. When this infection has once occurred, natural cure is almost hopeless.

OPERATIVE TREATMENT is indicated generally when:—

Adequate local rest must be secured, e.g., fixation of the spine or of a joint by a bone-graft.

The local focus is causing danger to life, e.g., tuberculous disease of the brain.

The local focus is likely to infect other important organs, e.g., tubercle of one kidney or of one testis.

The local focus affects an unimportant organ, e.g., tuberculous glands of the neck.

Tuberculous suppuration has occurred.

Pyogenic infection has occurred.

Other serious visceral disease is absent.

Rest and constitutional treatment have failed to cure.

INJECTION OF TUBERCULIN.—Various preparations from cultures of tubercle bacilli have been used for the treatment of the disease. The bacilli are killed by various thermal or mechanical means and an extract made from their remains; this forms tuberculin. The first dose varies from 0·00001 mg. to 0·0001 mg. according to the age of the patient, and is increased up to 0·001 mg., being given about once a week for a course of three months. Opinions differ much as to the value of these injections.

Diagnosis.—In obscure cases the following methods have been advised. They are not reliable, since even well-healed lesions, or some insignificant and quiescent glandular infections, will give the reaction. The reaction is related to anaphylaxis, and is not altogether devoid of danger.

Injections of the Old Tuberculin.—Koch's tuberculin (0·005 c.c.) is injected hypodermically. A sharp reaction follows in a few hours—malaise, temperature of 101°–104° F., and local pain and swelling of the affected focus. This is not free from danger, and is now seldom used except in the case of cattle.

Injections of the New Tuberculin (T.R.) in doses of 0·001 to 0·002 mg. This affects the opsonic index in tuberculous cases. Thus: (1) A marked alteration in the opsonic index after tuberculin; (2) A variable opsonic index when taken on different occasions; (3) A considerable rise in the index after massage or exercise—all indicate tuberculosis.

Tuberculosis—Diagnosis, ***continued.***

CALMETTE'S OPHTHALMO-REACTION.—A drop of a watery preparation from the dried T.R. is instilled into the eye. Within six hours slight inflammation of the conjunctiva, especially at the inner canthus, indicates a positive diagnosis. It is not free from a slight risk of causing ulceration or severe conjunctivitis.

VON PIRQUET'S CUTANEOUS REACTION.—Four scarification marks are made upon the patient's skin, and into two of these the new tuberculin is rubbed. The scratches thus treated show a marked inflammatory reaction within two or three days as compared with the others. This reaction, on account of its simplicity and freedom from untoward results, is now used almost to the exclusion of the other methods. Of value in children up to the age of 12 years.

ACTINOMYCOSIS

Aetiology.—It is caused by infection by the *Streptothrix actinomyces.*

THE ACTINOMYCES is an anaerobe and grows in cultures in the form of long branching filaments. In the tissues it is arranged in the form of radiating club-shaped masses, from which it was named the ray fungus (*Fig.* 20). These masses form granules in the pus which are visible to the naked eye. An aerobic form may occur. Filaments are Gram-positive, but the clubs are Gram-negative, and are only found in the body and not in cultures.

IN CATTLE commonly affects tongue and jaw, forming chronic hard swellings, which break down to form suppurating sinuses.

Distribution in Man.—

THE UPPER OR LOWER JAW, tongue, or floor of the mouth is attacked most often, by direct infection by diseased corn. About 55 per cent of cases occur in the region of the angle of the jaw, e.g., cervico-facial type.

THE LUNGS AND PLEURÆ are infected by inhaled particles, and chronic lesions like those of tubercle are caused. These may form an empyema, and the ribs and chest wall become the seat of chronic suppuration.

Any part of the alimentary tract, especially the CÆCUM, APPENDIX, and LIVER, may be infected by swallowed particles.

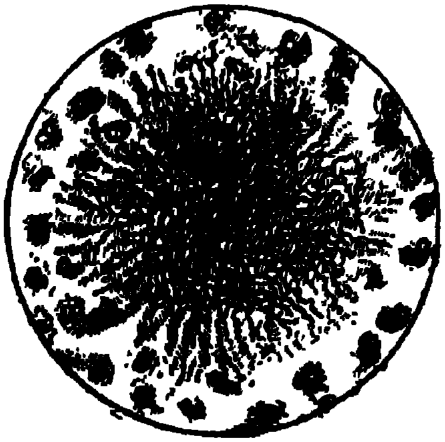

Fig. 20.—Mycelial filaments of actinomycosis surrounded by pus cells.

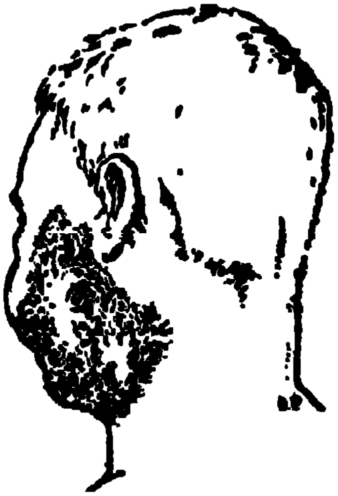

Fig. 21.—Actinomycosis. Dusky brawny thickening. Multiple sinuses.

Pathological Anatomy.—At first a hard, indolent nodule is formed, e.g., in the jaw. Usually at the site of a carious tooth. Then the soft parts are involved in the nodular mass, which softens and breaks down, forming a multilocular abscess (*Fig.* 21). This discharges pus containing the characteristic granules. The whole process is marked by pronounced fibrosis, producing a dense scarring, which, while it deforms the part, tends to limit the diffusion of the disease, which is therefore very chronic in its course. Secondary septic infection of the diseased areas occurs sooner or later, and is the ultimate cause of death.

Treatment is by local excision and erasion where possible, and by large doses of potassium iodide, ʒij thrice daily, or iodine in milk. If sinuses present these are scraped and laid open and packed with zinc peroxide paste (freshly made). Sulphapyridine therapy has proved useful in those cases where iodides have proved ineffective. Collosol copper injected along the edge of the indurated area has given beneficial results. X rays and radium are said to have curative effects. As a rule the visceral disease is surely though slowly fatal.

Madura Foot is an actinomycosis or mycotic infection of the foot. Only found in India and tropical regions, affecting bare-footed natives.
Chronic suppuration with multiple sinuses.
Destruction of the tarsal bones and joints.
TREATMENT.—Amputation.

CHAPTER VIII

TUMOURS AND CYSTS

Definition of Tumour.—An autonomous, heterogenous new formation of cells which do not obey the ordinary laws of physiological growth and subserve no useful function.

Adami suggests classifiying tumours histologically into two classes:—

1. BLASTOMATA.—Produced by abnormal growth of component cells of the individual.
    a. Pulp tumours: (i) Simple, e.g., fibroma, lipoma etc.; (ii) Malignant.
    b. Rind tumours (from covering and lining tissues): (i) Simple, e.g., papilloma; (ii) Malignant, e.g., carcinoma and endothelioma.
2. TERATOMATA.—Composed of cells of one individual included in the tissues of a second individual. These tumours arise from totipotent cells.

CLINICAL CLASSIFICATION

I. **Innocent Tumours.**—

Encapsuled, or if diffuse they do not infiltrate.
Do not affect the lymph-glands.
Do not recur after complete removal.
Do not disseminate.
Do not endanger life, unless they mechanically interfere with some vital organ, or unless they lead to hæmorrhage or infection.
Often multiple, and of different genera.

II. **Malignant Tumours.**—

Are not encapsulated, but infiltrate the surrounding tissues. Sarcomata are often in a capsule in their early history, but inevitably break through the capsule before long.
Affect adjacent lymph-glands.
Tend to recur after removal.
Become disseminated in distant organs.
Untreated they inevitably destroy life.
Primary growth is always single, except for rare cases of bilateral sarcomata.

THEORIES OF THE NATURE OF MALIGNANCY.—

1. COHNHEIM'S 'INCLUSION THEORY'.—Supposes the accidental inclusion of groups of embryonic cells in abnormal positions. These may be from cell 'rests', e.g., accessory spleen, pancreas, or adrenal, or from the vestiges of structures which have only an embryonic existence, e.g., branchial clefts.
2. THEORY of MICRO-ORGANISM OR VIRUS.—
    Facts in favour:—
    a The general similarity to bacterial diseases, the local origin, the dissemination, and cachexia.

b. The relation to some toxins of known bacterial origin, e.g., those of erysipelas.

c. The phenomena of Jensen's mouse cancer and Rous's chicken cancer, the cells of which can be inoculated from one animal to another.

Facts against:—

a. The most laborious research has failed to demonstrate an organism whose injection produces the disease.

b. The fact that metastatic growths consist of the same cells as the primary shows an exuberant activity in the tissue cells, and not merely that they are invaded by a foreign organism.

c. The great rarity of infection or contagion.

3. THE THEORY OF ALTERED CELL EQUILIBRIUM.—As the result of some stimulus the cells have the normal relations between function and reproduction altered so that they reproduce themselves indefinitely without regard to physiological needs. They destroy neighbouring cells and migrate to other organs, where they continue to proliferate, i.e., they become parasitic.

Facts in favour:—

a. The phenomena of conflict between the cancer cells and those in their vicinity.

b. The resemblances between the cancer cells and those whose chief object is reproduction, viz., the germinal cells, shown by the method of nucleus division.

HISTOLOGICAL CLASSIFICATION

I. BLASTOMATA

A. Simple or Typical Pulp Tumours.—

1. LIPOMA (*Fat Tumour*).—

SUBCUTANEOUS.—May be sessile or pedunculated.

Lobulated and encapsulated, and painless—the common kind.

Multiple, painful—adiposis dolorosa (Dercum's disease). These are neurolipomata.

Diffuse—in neck, axilla, groin.

Fatty hernia of the linea alba.

SUBSEROUS.—In peritoneal cavity—In hernial sacs—Exaggerated appendices epiploicæ—In spermatic cord.

SUBMUCOUS.—Rare—Small—Conjunctiva, larynx, and any part of alimentary canal.

SUBSYNOVIAL.—Forming pedunculated masses protruding into joint cavities—'Lipoma arborescens'.

INTERMUSCULAR.—Comparatively common.

INTRAMUSCULAR.—Rare.

PERIOSTEAL.—Mixed with striped muscle fibres, generally congenital.

MENINGEAL.—Either intra- or extra-dural—Often associated with spina bifida—Often congenital.

2. CHONDROMA (*Cartilage Tumour*).—Often degenerate into mucus, or ossify—Long bones of limbs at epiphysial junction—Especially from phalanges and in rickets. Sometimes affect the pelvis or sternum or spine, when they are likely to become chondro-sarcomata in later years.

ECCHONDROSIS.—Local outgrowth of articular cartilages, especially knee;

Simple or Typical Pulp Tumours—Chondroma, ***continued.***

Enchondroma.—Usually in the small bones of the hand and feet in young people. Ollier's disease (hereditary deforming dyschondroplasia) is a rare form of true enchondroma.

3. OSTEOMA (*Bone Tumour*).—

Compact or Ivory.—Dense and sessile. From frontal bones, especially in sinus or orbit—External auditory meatus—Angle of mandible.

Cancellous.—Tipped with cartilage until growth ceases. Generally from epiphysial lines of long bones.

Other Bony Outgrowths.—Subungual exostosis, generally from great toe—Ossified tendon insertions—Ossified muscles.

4. ODONTOMA (*Tumours developing from Teeth or Teeth Germs*).—

Epithelial.—Probably malignant. Epithelial columns with cystic dilatation embedded in fibrous tissue. Arise from enamel organ. (*See* Chap. XXVII.)

Follicular.—A follicle containing a permanent tooth crown remains deep in the jaw and becomes distended like a cyst. (*See* Chap. XXVII.)

Radicular.—Hard mass of dentine and cement attached to root of tooth.

5. FIBROMA (*Fibrous-tissue Tumour*).—

Simple.—Composed of masses of fibrous tissue. Gums (epulis)—Ovary—Uterus—Nerves.

Molluscum Fibrosum.—Overgrowth of skin and subcutaneous tissue in pendulous folds or pedunculated tumours. Often associated with multiple neuromata.

6. MYXOMA (*Mucoid Tumour*).—Very rare in pure form. Often present as degeneration product of other tumours. Nasal polyp (the common instance) is probably only œdematous granulation tissue.

7. GLIOMA.—Consists of delicate, branching, neuroglial cells. Brain—Spinal cord.

8. NEUROMA (*Nerve Tumour*).—

True.—

Multiplication of Actual Nerve Elements.—Rare.

Ganglioneuroma.—Ganglion cells and nerve-fibres usually associated with sympathetic cord.

Neuro-blastoma.—Less differentiated.

False.—Fibromata growing in nerve-sheath.

Localized.—Painful nodule in subcutaneous nerve.

Diffuse.—Molluscum fibrosum and Recklinghausen's neurofibromatosis.

Plexiform.—Myxomatous thickening, generally on nerves of scalp.

Amputation Stump Neuroma.

9. MYOMA.—Consists of unstriped muscle and connective tissue.

Occurs in uterus, bladder, œsophagus, and intestine.

Hard or soft, according to proportion of fibrous tissue to muscle.

Generally multiple and encapsuled.

Tends to degeneration and other changes: Calcification—Myxomatous degeneration—Gangrene—Sarcomatous growth.

Striped muscle tumour—rhabdomyoma—very rare. Congenital. Site: heart and kidneys.

10. OSTEOCLASTOMA OR GIANT-CELLED TUMOUR OF BONE.—
Consists of red marrow tissue, i.e., many multinuclear cells embedded in mass of round and spindle cells.
Very vascular, even pulsating.
Occurs in ends of long bones (especially head of tibia and lower end of radius), in mandible or maxilla, or as an "epulis".
Expands outer layer of bone to thin shell.
Does not disseminate nor affect lymph-glands.
Does not recur if removed locally.
MULTIPLE MYELOMATOSIS.—This condition is not to be confused with the single osteoclastoma. Multiple myelomatosis is a diffuse tumour-like affection, chiefly of spine, sternum and ribs, and skull, though may be any bone. Associated with Bence-Jones proteinuria. Radiosensitive, unlike single osteoclastoma.

B. Malignant or Atypical Pulp Tumours.—

1. SARCOMA (*Malignant Pulp Tumour*).—Occurs in any tissue of the body, bones being specially liable. Femur, tibia, scapula, and innominate are most frequently affected.
Very vascular. May cause a bruit.
Liable to infect blood-stream by venous dissemination, and thus cause metastatic growths, most commonly in lungs and liver.
Lymph-vessels are absent or scarce. Many varieties never infect lymph-glands.
Degenerative processes, hæmorrhagic or myxomatous, common.
Often occur in childhood or infancy.
In the case of the eye, kidney, ovary, and adrenal in infancy, the sarcoma may be bilateral.
Consists of immature connective tissue.
Differs from innocent connective-tissue tumour by preponderance of cellular over intercellular elements and the immature character of the cells.
Differs from malignant epithelial tumour by fact that the cells are not arranged in groups, and that they are separated by intercellular tissue and blood-vessels.
VARIETIES.—
a Round-celled Sarcoma.—Universal distribution in tissues, and occurs at any age.
Intercellular substance reduced to a minimum.
b. Lymphosarcoma.—Tissue with structure of lymph-gland—definite intercellular network enclosing small round cells.
c. Spindle-celled Sarcoma (including the so-called 'mixed-celled sarcoma').—Cells are fusiform, or oat-shaped. In sections some are round, others elongated.
Intercellular tissue often develops into fibrous tissue, cartilage, muscle, or bone.
Occurs in periosteum and in glands, e.g., ovary, testis, mamma, kidney, parotid.
In some the cells develop to resemble fibrous tissue, giving the fibrosarcoma (recurrent fibroid). Rarely form metastases, but tend to recur on removal.
d. Alveolar Sarcoma.—Cells arranged in alveoli like those of a carcinoma.
Originate in skin in hairy moles.

Malignant or Atypical Pulp Tumours—Sarcoma—Varieties, ***continued.***

e. *Melanoma.*—These are pigmented tumours, the simple type represented by the pigmented nævi, but from these and from tissues containing melanin, the intensely malignant melanoma may arise. Contains masses of brown pigment between and in the cells—Cells vary in size and shape—often of an alveolar type.

The urine may contain the pigment and blackens on exposure or after oxidation.

Originates in skin (moles), nail matrix, vulva, anus, palate, and uveal tract. At any age.

Intensely malignant. Infects lymph-glands and rapidly disseminates.

Note.—The precise classification of the melanomata is under dispute. Some authorities group with carcinomata, some with sarcomata.

f. *Chloroma.*—A lymphosarcoma of grass-green colour. Occurs in children and young adults, growing on the skull bones chiefly. It is intensely malignant.

TREATMENT OF SARCOMATA.—

Free and Early Removal of the affected part is the rule. In the case of round-cell sarcoma, melanoma, and sarcoma of the femur it is almost hopeless.

Radiation.—Some sarcomata are radio-sensitive (lympho-sarcoma). The tendency is for the tumour to disappear after radiation and then to return after an interval in a more radio-resistant form (if dissemination has not already taken place.)

Injection of Coley's Fluid.—An attack of erysipelas has been observed to check the growth of sarcomata. The mixed toxins of streptococcus of erysipelas and *Bacillus prodigiosus* are injected—if possible into the tumour—beginning with small doses and increasing until a rise of temperature is produced. Repeated daily or on alternate days for at least four weeks. If no improvement occurs in this time it is discontinued. If the case improves, continue for a year. Used also as a prophylactic against recurrence after operation. Best results have been with spindle-celled sarcoma.

2. ENDOTHELIOMA is a neoplasm which arises from endothelial tissues, usually in a gland, e.g., the parotid or testis. It includes a quantity of glandular tissue, together with cartilage, and is very prone to myxomatous change. Also develops in the serous membranes—pleura and peritoneum—and from the 'carotid gland' in the neck. The arrangement of the endothelium in branching columns gives a strong resemblance to an epithelioma.

ORIGIN may be from endothelium:—

Lining lymphatic clefts or spaces,
Lining blood- or lymph-vessels, or
Lining lymph-vessels in sheath of blood-vessels (perithelioma).

MENINGIOMA, sometimes termed a PSAMMOMA or ENDOTHELIOMA, arises from the endothelial cells of the arachnoid villi.

Cells are arranged in whorls. Central portions become calcified.

3. ADRENAL TUMOUR OR HYPERNEPHROMA.—Atypical carcinoma arising in renal tubules, which may be situated beneath renal capsule, beneath hepatic capsule, or in the pelvis.

Imitate the structure of the adrenal zona fasciculata.

Often occur in infants, and are then generally bilateral.

Metastases occur easily, spreading by the veins.

C. Simple or Typical Rind Tumours.—

1. PAPILLOMA.—Consists of a central axis of vascular fibrous tissue surrounded by layer of epithelium. Epithelium never dips down into the tissue below the basement membrane.

Note.—True papillomata are prone to carcinomatous transformation, and hence they are dangerous tumours, though benign at first.

WARTS.—Occur on the skin, tongue, or larynx.

Single and stationary: May develop into horn, sarcoma, or epithelioma.

Multiple: Possibly infective—Especially in children—Often vanish rapidly.

Genital: Result from vaginal or urethral discharges.

VILLOUS PAPILLOMA.—Delicate branching filaments occur in bladder or renal pelvis, and choroid plexuses of brain.

INTRACYSTIC PAPILLOMA —Occurs in ducts of glands, e.g., mamma or ovary.

2. ADENOMA.—Innocent tumour Consists of gland tissue unconnected with ducts. Often encapsuled. Often of mixed nature, e.g., fibro-adenoma.

May occur in any gland. Common in mamma, ovary, parotid, thyroid, sebaceous glands, and uterus.

Alveoli, having no duct outlets, are very often cystic.

When growing from a mucous surface they are pedunculated and called polypi. Common in rectum and uterus.

Usually occur in adolescence.

Note.—The true adenomata, while benign at first, are prone to carcinomatous transformation

3. ANGIOMA (*Tumour consisting of Blood-vessels*).—

SIMPLE NÆVUS —Skin, subcutaneous tissue, mucous membrane.

Birth mark, or port-wine stain: Involves only capillaries.

Telangiectasis: Arterioles, venules, and capillaries.

Nævo-lipomata. Encapsuled masses of fat and nævoid tissues

CAVERNOUS NÆVUS.—Skin, mucous membrane, surface of liver.

Tissue similar to erectile tissue, containing large blood spaces.

PLEXIFORM ANGIOMA.—Subcutaneous tissue, especially scalp or limbs.

A superficial mass of tortuous veins is a marked feature, and hypertrophy of the tissues is common.

Cirsoid aneurysm—Aneurysm by anastomosis, etc

4. LYMPHANGIOMA (*Tumours consisting of Lymph-vessels*).—

SIMPLE OR CAVERNOUS.—Dilated lymphatics. In skin, mucous membrane, especially tongue or lower lip.

LYMPHATIC CYST.—Cystic hygroma—Usually congenital—Neck, axilla, or groin.

D. Malignant or Atypical Rind Tumours.—

1. CARCINOMA.—Malignant tumour. Consists of mass of epithelial or gland tissue of an abnormal type. Chief points are:—

Arise in situations where the tissues are exposed to constant irritation (chemical or possibly mechanical), e.g., the tongue of clay-pipe smokers, on the skin of those who work with tar, paraffin, soot, or X rays. Also in the site of ulcers or scars, e.g., in stomach, cervix uteri, or lupus scars.

Glandular alveoli penetrate below basement membrane.

Alveoli may consist of solid columns of cells instead of hollow tubes.

Irregular and disorderly proliferation of gland tissue.

Malignant or Atypical Rind Tumours—Carcinoma, ***continued.***

The fibrous tissue in the neighbourhood undergoes a marked proliferation which is of the nature of a hostile reaction.

They are rich in lymphatics and poor in blood-vessels.

Very liable to ulceration, necrosis, or colloid degeneration.

Quickly infect lymph-glands—Rapidly disseminate.

Local infectivity is shown by growth of a second tumour in a place in contact with the first, e.g., from one labium to the opposite, from an ulcerating cancer of the breast to the skin of the arm. These occurrences are very rare. In operations where the cancer is cut into, the stitch holes may be the seat of recurrent growth.

Very rare in young—Commonest at 45–65.

Bröder has classified carcinomata into four groups according to type of cell and its arrangement Group I least malignant, Group IV most malignant.

VARIETIES —

a. Squamous-celled Epithelioma.—Consists of downward growing columns of stratified epithelial cells, simple or branching.

Begins as a wart, ulcer, or fissure

Occurs in any surface covered by stratified epithelium—Mouth, tongue, œsophagus, larynx, anus, penis, scrotum, vulva, vagina, cervix—Skin, especially at site of warts, scars, or chronic ulcers —Bladder.

Columns of down-growing epithelium often become horny, forming cell-nests or epithelial pearls

Rapidly invades lymph-glands, and invades neighbouring structures. Visceral metastases are rare.

b. Columnar-celled or *Acinous.*—Arising from and mimicking a tubular gland structure.

Commonest in alimentary canal—œsophagus, stomach, colon, and rectum (*Fig* 22)—uterus.

c Spheroidal-celled —Solid columns of spheroidal cells

Commonest in breast (*Fig.* 23) and stomach

Scirrhus: A spheroidal-celled carcinoma in which a large proportion of connective tissue occurs, forming a very hard growth. Especially common in old patients.

Encephaloid Cancer: A carcinoma containing a minimum of connective tissue, forming very soft growths.

Usually spheroidal-celled. Especially common in young patients.

d Rodent Ulcer or Basal-cell Carcinoma (*Fig.* 24).—

Probably starts in the cells of sebaceous glands—Begins as a nodule on the skin—Ulcerates after several years.

Slowly destroys every tissue it meets, e g., eyeball, bones of face.

Edges show but little heaping up—Often covered by a scab, but never heals by cicatrix

Never infects lymph-glands Never disseminates.

Commonly occurs on nose, eyelids, orbital angles, or cheek. May occur on neck, or rarely on trunk.

Common in the aged, rare before 40

Seldom recurs after complete removal.

Microscopically, it contains no 'prickle' cells like an epithelioma, it never has cell-nests or any keratinization, and its cells are smaller than those of an epithelioma.

2 CHORION-EPITHELIOMA or deciduoma malignum —
Generally occurs in the uterus after a miscarriage
Consists of tissue like that of the early fœtal chorionic tissue, viz Multinucleated masses of protoplasm, the syncytium, and many spindle cells like those of Langhans' layer
Exactly similar tumours occur in the testis, therefore they are, strictly speaking, teratomata
Leads to early multiple visceral metastatic growths, especially in the lungs

The Treatment of Cancer

EARLY REMOVAL, with a good margin of healthy tissue and with the associated lymphatic area, is imperative in all possible cases

X RAYS —For superficial growths e g, rodent ulcer or recurrent nodules in the skin after breast cancer, X rays are often curative For certain deep growths, lymphosarcoma, lymphadenoma, or even metastases of carcinoma in bones, deep X rays are efficacious, causing temporary arrest or disappearance of the tumour and alleviation of symptoms Recently the low voltage contact X-ray therapy (Chaoul) has produced some good results in skin carcinoma buccal carcinoma, and has been tried in cases of carcinoma of the bladder

RADIUM —Is used as a potent source of radio activity Radium gives off three sets of rays α rays with no penetrating power β rays, with little

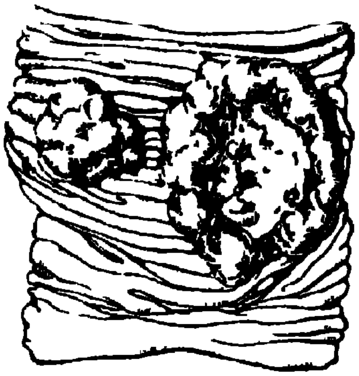

Fig 22 —Malignant ulcer of rectum

Fig 24 —Rodent ulcer

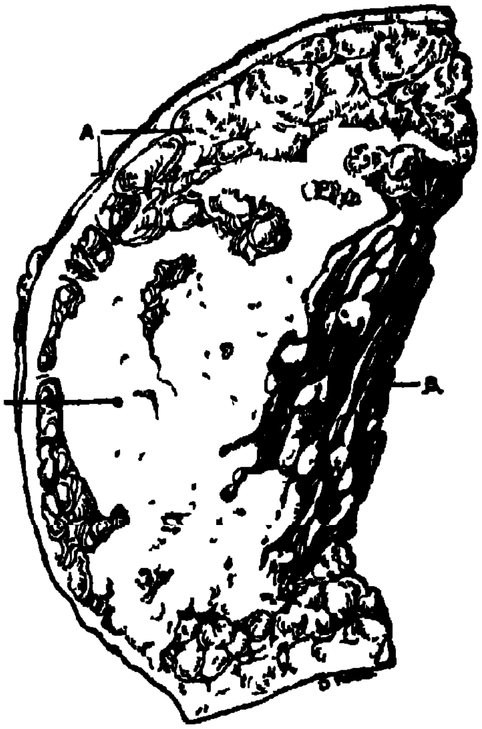

Fig 23 —Scirrhous cancer of breast invading skin and muscle ,A Breast tissue B, Muscle C New growth

Treatment of Cancer—Radium, ***continued.***

penetration but with destructive effect on the tissues (if unscreened radium is used, it will by reason of the β rays act as a caustic and make a deep burn very slow to heal); γ rays with great penetrating power and with a selective action on germinating cells, especially those of malignant growths. A solution of radium salt gives off a radio-active gas called radon, the potency of which lasts only for about one week.

FORMS OF APPLICATION.—Radium is employed in four ways: (1) *Flat applicators* containing about 5 to 20 mg.; (2) *Platinum needles* containing 1 to 5 mg in the form of one of the salts, usually the sulphate, the platinum case being about 0·6 mm. thick so as to screen off the β rays; (3) *Radon seeds*—glass or metal capsules containing radium emanation, each containing about 1 to 5 units of emanation (this unit is called a millicurie and is $\frac{1}{1000}$ part of the gas which would be given off from 1 g. of radium); (4) *A bomb* (teleradium)—a large quantity of radium, 4 to 5 g., in a heavy metal container, through a screened window of which the rays can be directed on to the patient from a distance of 1 to 6 ft. Severe constitutional symptoms are likely to arise during bomb treatment as the blood-count diminishes rapidly. Dosage is expressed in 'r' (Roentgen) units—the r-unit being that quantity of radiation which under standard conditions produces a conductivity such that the saturation current equals one electrostatic unit.

INDICATIONS FOR VARIOUS APPLICATIONS.—In most cases, the method of choice is *interstitial radiation*, by which needles containing 1 to 3 mg are buried in the tissues in and around the growth so that about 1 mg. of radium is allotted to each cubic centimetre of tissue, these are left in place for about 6 to 10 days. *Surface radiation* is by the flat applicators heavily screened by lead, or by incorporating a series of needles in rubber or wax so as to act at about 1 or 2 cm away from the skin; thus the whole of the front of the neck or the chest may be radiated so as to act upon glands or possible fugitive malignant cells. *Radon seeds* are used for inaccessible places, e.g., the brain; they are left permanently in place. *The bomb* is used in order to irradiate a wide area of deep lymphatics, e.g., in the chest or pelvis after a primary growth in the breast or uterus has been treated.

INDICATIONS AND RESULTS OF RADIUM TREATMENT OF MALIGNANT DISEASE.—It is impossible as yet to estimate the proper value of radium, because there has not been time enough to wait for late results. Moderate opinion would accept the following estimate. In suitable cases, radium has the same effect as excision of a primary growth, with the advantage of avoiding a mutilating and sometimes dangerous operation. It can often arrest for a long time an inoperable growth. It is doubtful whether it can prevent or cure lymphatic spread.

Superficial Growths such as Rodent Ulcer or Secondary Skin Nodules after Cancer of the Breast.—Radium is effective. But in rodent ulcer, if the effect is not immediate and complete, it is better to excise.

Mouth.—Epithelioma of the tongue or lips, and some cases of laryngeal growth, should be treated by interstitial radiation, followed by excision of the glandular areas. The growth disappears, mutilation is avoided, and mobility of the parts is restored. There is no evidence that late results are better than after excision.

Cervix Uteri.—Radium has given such good results that it has been adopted as the routine treatment by many surgeons. It avoids the ordeal and high mortality of the radical Wertheim operation, but it is too soon to say whether the late results are as good.

Breast.—Radium is indicated in all inoperable cases or those with involvement of skin and glands, and in patients who refuse operation. Only a few enthusiasts use it for early cases, where radical operation gives such good results.

Must irradiate:—

1. Whole of breast.
2. Axillary lymphatic glands.
3. Supraclavicular lymphatics
4. Parasternal lymphatic glands.

Brain, Rectum, Bladder.—In these the method is under trial and is a hopeful alternative to doing nothing in inoperable cases, but not justified in those where the growth can be removed.

Bone.—Radium is useless, because it cannot effectively penetrate the bone tissue without causing extensive necrosis.

VARIOUS PALLIATIONS.—From time to time different injections have been tried in hopeless cases, with an occasional improvement. Substances such as colloidal lead, lead selenide, goat's serum, extracts of spleen, pancreas, placenta, and the like have all been tried.

E. Dermoids.—Skin or mucous-membrane tumours occurring in situations normally devoid of these structures.

SEQUESTRATION DERMOIDS.—Occur in masses of embryonic epiblast which have become sequestrated in the deep tissues during development especially along lines of fusion of epiblastic folds.

SITUATION.—Mid-line of body, except from tip of nose to occiput. In facial and branchial clefts, inner and outer canthus of eye, sides of nose, angles of mouth, pinna of ear.

Over scalp in the cranium and root of nose they are caused by patches of epithelium being cut off by inward growth of membranous bones from the surface skin Hence these varieties either lie in a hollow of the bone or else underlie the bone altogether.

IMPLANTATION CYSTS.—Found under the skin or conjunctiva when pieces of epithelium have been buried by injury.

TUBULO-DERMOIDS.—Arise in a persistent fœtal duct

LINGUAL.—Upper part of the thyroglossal duct.

THYROGLOSSAL—Anywhere in mid-line from hyoid to sternum, from persistence or diverticulum of thyroglossal duct

BRANCHIAL.—Cysts beneath the deep fascia of neck arising from branchial clefts

RECTAL.—From post-anal gut. They may project into rectum as a polypus. They may lie between rectum and sacrum. They may bulge out behind as sacrococcygeal tumour

URACHAL.

OVARIAN DERMOIDS.—Probably arise from an ovum by an abnormal reproductive development, possibly by parthenogenesis or by inclusion of a fertilized ovum within the body of the fœtus.

Contain skin and glandular structures of the utmost complexity: hair (often very long); glands (sebaceous, sweat, mammary); skin appendages (horns, nails, epithelial pearls); bone and teeth.

Somewhat similar tumours occur in testis

Dermoids, *continued*

MOLES.—Dermoid patches. Hairy, pigmented, raised areas of skin or conjunctiva.
Base consists of large cells arranged in an alveolar fashion.
Often form starting-place of melanotic tumours

II. TERATOMATA

A teratoma is a conglomerate mass of fœtal tissues and organs.
Represents a 'parasitic' fœtus, that is, an incompletely separated and ill-developed twin.
Or may possibly arise from activity of an undifferentiated reproductive cell.
Commonest in sacrococcygeal region.
Ovarian and testicular dermoids may be of this nature.

CYSTS

Of Embryonic Origin.—
DERMOID CYSTS (*see* p. 69).
DENTAL CYSTS (*see* ODONTOMA, Chap. XXVII)
TUBULO-CYSTS —Dilatation of functionless ducts.
CYSTS OF THE URACHUS, CYSTS OF VITELLO-INTESTINAL DUCT.
CYSTIC DISEASE OF TESTIS and CYST OF THE EPIDIDYMIS arise from Wolffian gland and duct
PAROÖPHORITIC CYSTS PAROVARIAN CYSTS, and PARAVAGINAL CYSTS arise from Wolffian tubules and duct in female
CONGENITAL AND INFANTILE HYDROCELES

Distension Cysts.—Formed by the distension of closed cavities.
GLANDULAR —Thyroid—Pituitary—Some ovarian.
NEW GROWTHS, especially adenomata —Adenoma of breast—Ovarian adenoma.
PERITONEAL —HYDROCELE—Tunica vaginalis—Canal of Nuck—Funicular process—Ovarian—Hydrocele of hernial sac
SYNOVIAL.—
BURSÆ.—Distension of normally-placed bursæ, e g., prepatellar—Bursæ connected with joints, so-called 'Baker's' cysts—Bursæ of new formation, e g , that between hyoid bone and thyroid cartilage.
GANGLIA.—Hernia of synovial membrane through a tendon sheath.
VASCULAR SPACES or extravasation —
BLOOD CYSTS.—Hæmatocele—Breaking down of vascular new growths —Arachnoid cyst
LYMPH CYSTS.—Hygroma—'Hydrocele' of neck—'Serous' cysts of mamma, axilla, or groin
NEURAL CYSTS —
Hydrocephalus · distension of ventricles—Syringomyelia: distension of central canal of spinal cord—Meningocele, etc. · spina bifida.

Retention Cysts.—Formed by a blocking of gland ducts.
Galactocele—Cystic disease of breast—Ranula—Dacryops—Pancreatic cyst—Hydrometra—Hydronephrosis—Hydrosalpinx—Hydrocholocyst.

Parasitic Cysts. —Echinococcus (hydatid cysts), Cysticercus.
HYDATID CYSTS.—
DISTRIBUTION.—In Australia and the Arctic regions, where dogs are very intimately associated with human beings, the disease is common; elsewhere it is rare.

The Parasite is a small tape-worm, the *Tænia echinococcus*, the adult form inhabiting the intestine of dogs and other animals, and the embryonic form growing in human tissues inside cysts.

Life History.—The head consists of a worm half an inch long, with four segments. The head has four small suckers and a row of hooklets. The posterior segment is the largest and is filled with the genital organs. From these the ova are discharged, and are then,by contamination with food, water, or uncooked vegetables, conveyed to the human host. The embryo develops in the stomach or intestine, and has four suckers and a set of hooklets It burrows its way into the blood-stream, and is deposited in the liver or any other tissue. Here it forms a hydatid cyst.

The Hydatid Cyst consists of two layers: an ectocyst, which is firm and chitinous, and an endocyst, which is protoplasmic. The tissues of the host form a firm fibrous layer or capsule outside the cyst. The endocyst is formed by germinal protoplasm, from which generally grow: (1) Numerous daughter cysts; (2) Brood cysts containing other embryos in their interior; (3) Solitary heads of mature worms known as scolices. The fluid in the cyst is of specific gravity 1007, with a trace of albumin, some sodium chloride, and a quantity of free hooklets, by which the nature of the fluid is commonly recognized.

Variations.—If the cyst contains no brood cysts, daughter cysts, or scolices, it is known as barren, or an acephalocyst. Sometimes the main cyst disappears and a collection of daughter cysts and scolices lie free in the tissues. This is the common condition in hydatid disease of bones, and is known as exogenous development.

Situation of Cysts—Liver, brain, kidneys, bone, or any other tissue, the first named being much the commonest.

Results.—

1. Simple growth, causing pressure symptoms.
2. Rupture into a serous cavity, causing toxæmia and marked urticaria. The scattered scolices may embed themselves and grow in the peritoneum or pleura.
3. Death of the parasite. The cyst then shrivels up, and may eventually become caseous or calcified
4. Suppuration, with all the possible eventualities of an abscess, e g, septic absorption, rupture, etc.

Diagnosis—Certain laboratory tests are of use in diagnosis of hydatid disease —

1. Precipitin test between patient's serum and hydatid fluid. Accurate in 65 per cent of cases.
2. Complement-fixation test.
3. Intradermal test (Casoni's reaction)—an urticarial wheal is raised as a result of introduction of hydatid cyst fluid intradermally.
4. Eosinophilia is present

CYSTICERCUS CELLULOSÆ is the intermediate or embryonic form of the common tape-worm or *Tænia solium*. The cysticercus stage is generally found in the flesh of pigs, and the adult tænia in the gut of man. But sometimes, especially in Germany, the cysticercus develops in human tissues as one or many cysts of varying size. The commonest situation is the brain or eye, but occasionally it may form a cyst the size of a pea under the skin. In the brain they are eventually fatal. They cause destruction of the eye. From the skin they should be excised.

CHAPTER IX

WOUNDS

Definition.—A forcible solution of continuity of the soft tissues.

Contusions.—Skin is not broken.

ECCHYMOSIS OR BRUISING produced by rupture of small blood-vessels.

If blood is poured out in some quantity, and enclosed by fascia or membranes, a

HÆMATOMA results Fluid at first—Becomes hard when coagulation occurs—This hardness begins as a peripheral ring—Results in: absorption, organization into a fibrous mass, serous cyst (e.g., arachnoid cyst), or suppuration.

TREATMENT.—Evaporating lotion, or hot fomentations. Firm pressure Aspiration. Aseptic incision as a last resort in very exceptional cases.

Incised Wounds.—Clean cut, with little or no bruising. Wide gaping of lips. Free hæmorrhage.

TREATMENT —

1. ARREST HÆMORRHAGE
2. RENDER PARTS AS ASEPTIC as possible Explore for nerve or tendon injury or injury to important viscera or ducts. Excise edges
3. Sew margins together
4. DRAIN only if Deep infection cannot be got rid of—Deep bleeding cannot be arrested—Pressure cannot be applied (e g , neck)—Wound communicates with deep cavity where extravasation exists, e.g , wounds of loin.
5. DRESS, with efficient application of
6. PRESSURE.
7. ATTEND TO THE GENERAL HEALTH Antitetanus serum in road or field cases.

FAILURE TO UNITE by first intention is due to.—

1. INEFFICIENT ARREST OF HÆMORRHAGE.
2. INEFFICIENT ASEPSIS, or foreign body in wound
3. INEFFICIENT SUTURING.
4. ABSENCE OF DRAIN in cases mentioned.
5. INEFFICIENT DRESSING, i.e , infection after sewing up.
6. GENERAL DISEASE.
7. CONSTITUTIONAL DEBILITY.

IF INFECTION HAS OCCURRED it is indicated by:—

Rise of temperature, rigor, etc.—Local pain and throbbing—Signs of inflammation round wound.

TREAT by: Opening freely—Washing out with peroxide of hydrogen—Freely draining.

DEATH MAY OCCUR from shock, hæmorrhage, or septic infection.

Lacerated and Contused Wounds.—

Produced by a blunt instrument, which tears the soft parts.
Wound is associated with injury to the surrounding parts.
Hæmorrhage is slight, or absent, as vessels have been torn rather than cut.
Edges torn and bruised, with little or no gaping.
Parts may be cut off from their blood-supply by—original injury, inflammatory reaction (thrombosis), or pressure of bandages.
Hence SLOUGHING and GANGRENE are common occurrences.

IF ASEPTIC it may heal by first intention if edges can be brought together, but generally some sloughs have to be absorbed or cast off, and healing is by granulation.

IF SEPTIC—as the majority of such cases are—acute inflammation, resulting in suppuration, sloughing, or gangrene, attacks parts round the wound.

This secondary sloughing is responsible for the loss of much more tissue than is the original injury in most cases.

After sloughs have separated, wounds heal by granulation

DEATH may occur from shock or septic infection.

TREATMENT.—

Antitetanic serum—prophylactic dose of 500 units. Sulphonamides by mouth.

Clean up (under anæsthetic if necessary). Trim off ragged edges and remove parts likely to slough Local application of penicillin.

Leave wound open. or delayed primary suture. if excision of wound cannot be satisfactorily and completely performed. Trueta's closed plaster technique is of great value in the treatment of extensive wounds of the soft parts of this type. All damaged tissue is excised and the wound packed lightly with sterile gauze or vaseline gauze and the whole area encased in plaster and thus immobilized. Discharge of pus occurs into the plaster Healing of the wound proceeds under the plaster. The plaster requires changing at intervals depending on the amount of discharge from the wound and the general condition of the patient

If septic inflammation occurs—Hot fomentations until sloughs have separated or suppuration has ceased.

If wound is very severe and involves a limb—

AMPUTATION WILL BE CALLED FOR: If limb has been torn off—If part is quite disorganized—If gangrene occurs, or is imminent—If severe septic symptoms occur—In severe crushes of the foot in elderly patients.

AMPUTATION WILL BE NECESSARY in the following conditions *if they result in gangrene, but not otherwise* · Compound comminuted fracture—Extensive stripping of soft parts from bone—Wound of the main artery.

THE NECESSITY FOR AMPUTATION IS INCREASED BY: Age and debility of patient—Distance from heart and bad blood-supply (hence the foot is the most likely part to require amputation)—Severe sepsis.

Crush Syndrome.—A clinical condition found in people who have been pinned under collapsed masonry during air raid. There is marked shock which goes on to anuria

Crush Syndrome, *continued.*

TREATMENT.—A high fluid intake is necessary—6 to 7 pints in the twenty-four hours. Sodium citrate, 2·5 g. and sodium bicarbonate, 2·5 g., should be given every three hours to keep the urine alkaline. Decapsulation of the kidney may be required as a last resort.

Punctured Wounds.—Long deep wound with small orifice.

LIABILITY TO INJURE DEEP STRUCTURES:—Blood-vessels—Nerves—Viscera—Serous cavities—Joints.

If infected, deep suppuration occurs.

OFTEN LEAVES A FOREIGN BODY in depth of wound.

TREATMENT.—

1. In the absence of evidence of sepsis or symptoms due to injury to deep structures—Simply clean and dress.
2. If deep structures give symptoms of injury—Open depths of wound and deal with deep structures
3. If there is any question of foreign body—Locate with X rays. Remove only if it is causing, or likely to cause, symptoms.
4. If suppuration occurs—Enlarge wound and drain

Gunshot Wounds.—

FROM MODERN RIFLE BULLETS.—

Simple punctured wound.

Straight track between the aperture of entry—small round puncture—and aperture of exit—a small slit

Only injures structures in its direct path

Sepsis, or introduction of foreign bodies, is rare

SOFT-NOSED BULLETS, dum-dum bullets, and bullets of low velocity produce much more laceration from their disruptive force.

DEEP INJURIES.—

BLOOD-VESSELS.—Fatal hæmorrhage—Traumatic aneurysm (when artery has been button-holed)—Arteriovenous aneurysm.

BONES.—Shaft Compound comminuted fracture—Cancellous tissue may be simply perforated.

HEAD.—Splintering of the inner table—Destruction of cerebral substance along the track of the bullet—Not necessarily fatal.

ABDOMEN.—If the gut is empty its perforation closes by a local plastic process without general peritonitis.

Liver, spleen, and kidneys recover without much damage

Hæmorrhage from injury to vessels is most common cause of death.

TREATMENT OF GUNSHOT WOUNDS.—

CLEAN THROUGH-AND-THROUGH BULLET WOUNDS.—Expectant treatment

SHELL WOUNDS.—Antitetanic serum, 500 units. Locate foreign bodies by X rays Excise wound in cone-shaped manner, with superficial and deep tissues which have been torn and infected. Remove foreign bodies and unattached bone fragments. Oral administration of sulphonamides

Primary suture in suitable cases dealt with within 12 hours of injury. This is now rarely employed in modern war wounds, it being better to proceed as below.

Delayed primary suture, i.e., inserting stitches at once and tying them within forty-eight hours if wound remains clean.

Secondary suture when cleaning only takes place by granulation.

For infected wounds, later than twenty-four hours, use one of the following methods:—

1. Hypertonic saline. Gauze wrung out of 10 per cent salt solution.
2. Carrel's tubes and hypochlorite solution.
3. Flavine and penicillin pack.

It is essential to obtain free and adequate drainage of these infected wounds.

Blast Injuries.—Although blast injuries are more common in the lungs and intestinal tract, yet some linear wounds of the face and trunk may be produced by the blast from exploding bombs. These fissured wounds should be powdered with sulphanilamide and covered with tulle gras.

Insect Bites.—

Bites by flies, fleas, bugs, lice, mosquitoes, etc., may result in two kinds of complications, viz :—

1 LOCAL INFLAMMATION WITH SEPTIC COMPLICATIONS —

Any type of cellulitis or boil at the point stung.

Frequently marked urticaria from irritant poison of the insect bite.

Œdema may be very marked if lip, tongue, or fauces are the seat of the bite.

Septicæmia may be caused by infection of the wound by pyogenic organisms

TREATMENT —That of any other infected wound

2 THE TRANSMISSION OF SOME SPECIFIC DISEASE of which the insect is the carrier—e g., malaria or yellow fever from mosquitoes, plague from rat fleas, etc

Stings of Bees, Wasps, Scorpions.—

The wound is infected with a definite irritant poison, and in the case of bee stings the sting remains in the wound Local irritation, inflammation, and œdema will be severe

TREATMENT.—Extraction of the sting by local incision will be necessary. Local application of alkaline lotions will neutralize the acid irritant

Snake Bites.—

Poisonous snakes are common in India, Australia, Africa, Central and South America

Three types of poisonous snakes are the cobra, the rattlesnake, and the viper, in this order of danger

The venom is the secretion of the parotid gland, squeezed into grooved fangs, the teeth of the upper maxilla.

Venom is a protein substance, the chief active constituents of which are a globulin and a peptone.

It is a virulent poison to all other animals except venomous snakes.

Animals, e.g., the horse, can be immunized by repeated small doses—the blood then contains an antibody From such blood the antivenene is prepared

The poison is destroyed by salivary and pancreatic ferments, and therefore is less dangerous when swallowed.

Snake Bites, ***continued.***

SYMPTOMS.—

SYMPTOMS DUE TO BLOOD CHANGES.—If injected directly into a vein, diffuse thrombosis with instantaneous death results. Otherwise destruction of blood-cells and of capillary walls results, with ecchymosis, multiple hæmorrhages, œdema, and local gangrene.

SYMPTOMS DUE TO NERVE PARALYSIS.—The medullary centres become paralysed. Speech centres, swallowing, and then respiratory and heart centres fail. Death usually occurs within 24 hours.

TREATMENT.—

LOCAL.—Tight ligature above bitten part. Incision into bitten area, with cupping (or sucking) of the wound. Injection into the tissues of a 1 per cent solution of calcium hypochlorite or chloride of gold. Failing these remedies, rubbing in solid potassium permanganate crystals.

GENERAL.—Alcohol, ammonia, and strychnine have been advocated, but they are of the same doubtful value as in the treatment of shock.

SPECIFIC —Injection of 20,000 units of antivenene. This is of most value for the cobra bite, and less so for that of the rattlesnake. The antivenene is polyvalent.

Methods of Healing of Wounds.—

HÆMORRHAGE fills interstices with clot

THROMBOSIS closes ruptured vessels.

EXUDATION of leucocytes and serum from thrombosed vessels into edges of wound and blood-clot.

ABSORPTION of blood-clot and dead tissues by the leucocytes

SUPPURATION —If the wound is infected with a few micro-organisms of a mild type, these too are absorbed by the leucocytes If, however, the infection is of a more virulent type, the leucocytes are killed, and form, with the serous exudation, pus.

FIBROBLASTS arise from endothelial cells and from flexed connective-tissue cells, and line the edges or surface of the wound They are large, elongated cells, with distinct nuclei.

VASCULARIZATION of the layer of fibroblasts is brought about by a budding of solid outgrowths from the capillary walls. These outgrowths eventually become canalized, and joining other channels, form new capillary loops.

GRANULATION TISSUE is thus formed by a layer of fibroblasts vascularized by new capillary loops.

SURFACE OF WOUND heals by growth of epithelial cells from the cut epithelial edges, over the summit of the granulation tissue.

CICATRIZATION of the wound is caused by conversion of granulation tissue into white fibrous tissue, the fibrillæ of which arise from the protoplasm of the fibroblasts, or as an intercellular exudation. Both nucleated cells and blood-vessels ultimately tend to disappear from the scar tissue owing to the contraction and compression of the fibrillæ.

SCARS OF RECENT WOUNDS will therefore be more vascular, i.e., redder, than surrounding parts in their early stages, when they consist of vascular granulation tissue. They will be less vascular, i.e., paler, than surrounding parts in their later stages when they consist of avascular fibrous tissue.

CUTICLE COVERING SCARS is devoid of any skin structure or appendages. No hair, glands, papillæ, or lymphatics.

Modifications of Healing.—

HEALING BY FIRST INTENTION.—

No gap is left between the divided wound surfaces.

No loss of tissue by sloughing or septic infection.

Granulation tissue is present as a thin layer only, between adjacent surfaces.

Scar consists of a mere line of fibrous tissue.

HEALING BY SECOND INTENTION, or healing by granulation.

A GAP IS LEFT between the divided edges of the wound, or

A GAP IS CAUSED by the loss of tissue due to sloughing or septic infection.

When the edges have been sewn up, the depth of the wound is occupied by the dead tissues, either sloughs or pus, and further healing cannot occur until these have been removed by the opening of the wound.

Small aseptic sloughs may be absorbed or digested by the subjacent granulation tissue.

Large aseptic sloughs will be simply cast off.

Septic sloughs will be separated by a process of acute inflammation involving the loss of substance of more or less of the living tissues in the edges of the wound.

THE GAPING FURROW between the wound surfaces has to be filled up by granulation tissue.

THE LARGE AREA OF GRANULATION TISSUE has to be covered over by an extensive sheet of epithelium.

The epithelium does not grow over the granulations unless the latter are on the same level as the edges of cut epithelial surface.

This 'levelling up' is produced by the gap being filled with granulation tissue, and also by the contraction of the latter drawing the edges and the base of the wound towards one another.

Epithelium grows slowly. If large areas are denuded of epithelium, skin grafting should be performed to prevent undue delay and excessive contraction.

THE SCAR resulting from this is a large mass of fibrous tissue in which much contraction takes place.

HEALING BY UNION OF GRANULATING SURFACES.—

Produced when a gaping wound is brought together after it has become covered with granulations.

Granulating surfaces will unite is sepsis is absent, and if granulating surfaces are healthy and vascular

HEALING UNDER A SCAB.—By granulation, when the granulating surface is covered by a mass of dried blood and exudation.

HEALING BY ORGANIZATION OF BLOOD-CLOT.—

Large spaces are left in the depth of the wound which become full of blood-clot.

The blood is removed by leucocytes, and these are replaced by granulation tissue.

Blood only acts as a passive scaffolding for the activity of leucocytes.

Modification of Scars.—

EXCESSIVE CONTRACTION produces great deformity when large subcutaneous areas are involved in the neck or flexures of limbs.

TREAT by dividing scar, stretching contracted part, and grafting new skin. Inlay grafts.

KELOID.—A vascular fibroid mass raised above the surface, and sending radiating processes in surrounding parts.

Specially frequent in tuberculous patients and negroes.

More often occurs when antiseptics are used than when these are avoided.

May be due to the involvement of a subcutaneous muscle (e.g., platysma) in the scar, constantly dragging upon it during healing.

Recurs on removal, but often disappears spontaneously.

TREAT by X rays or radium. Small scars excised completely and an inlay graft applied to the area

ULCERATION.—Due to defective blood-supply.

PAINFUL SCARS.—Due to implication of nerve terminals or a bulbous nerve-end by contracting scar.

TREAT by opening and freeing or excising nerve.

MALIGNANT DISEASE may attack any scar Generally affects superficial scars subject to much irritation, e.g , X-ray burns.

Slow-growing, fungating epithelioma.

Pain and lymphatic infection do not occur until neighbouring skin is invaded.

CHAPTER X

ASEPSIS AND ANTISEPSIS

The systems adopted to prevent wound infection have been variously termed Antiseptic and Aseptic.

The Antiseptic System aims at the destruction of bacteria, and relies largely upon the action of chemical bactericides, wounds themselves being treated with such chemicals. Instruments are prepared by soaking in, dressings by impregnation with, skin by scrubbing with, antiseptics. The air is treated by an antiseptic spray, and the wounds are constantly bathed in antiseptic solutions. This is Lister's surgical technique

The Aseptic System aims at the prevention of wound infection by excluding bacteria from the wound, relying almost entirely on heat for the destruction of germs, and using no chemicals in contact with wounded tissues. Instruments are boiled, dressings are steamed, skin surfaces are excluded from wound contact

The Prevention of Wound Infection.—All are agreed that there are three great factors in the prevention of wound infection, viz.:—

VITAL RESISTANCE of the living tissue.

EXCLUSION OF BACTERIA from contact with wounds

DESTRUCTION OF BACTERIA by artificial agencies

These three factors differ in their relative importance under different circumstances, thus:—

IN DEALING WITH HEALTHY TISSUES AND UNDER IDEAL CIRCUMSTANCES.—It is possible so far to exclude germs that the vitality of the tissues can be depended upon to prevent infection without the use of any chemicals.

IF THE TISSUES ARE ALREADY INFECTED.—E g., in dealing with an abscess. The exclusion of further infection is all that can be attempted, and it is useless to apply chemicals to the tissues to kill the germs *in situ*, because the vital resistance will be injured as much as the vitality of the bacteria.

WHEN INFECTION IS PROBABLE OR CONDITIONS ARE NOT IDEAL.—E.g., an amputation in the proximity of unhealthy tissues, or emergency operations in poor houses with inexperienced assistants The use of antiseptic solutions throughout the operation, both in bathing the wound and in saturating the towels which surround it, will be a great safeguard in preventing infection. The chemicals used are dilute, and serve rather to inhibit the action of bacteria than to kill them outright.

THE PREPARATION OF SKIN SURFACES is the most difficult and debatable factor in the whole subject. It is agreed that it is impossible always to render the skin aseptic or entirely to exclude its contact with the wound. Hence after all has been done to render it sterile, it is treated as an infective agent and excluded as far as possible.

THE PREPARATION OF DRESSINGS, LIGATURES, AND INSTRUMENTS.—Here all agree that sterilization by heat is the best method of routine, but the

The Prevention of Wound Infection, ***continued.***

use of chemical antiseptics finds a place under certain circumstances (*see below*).

THE USE OF SPECIAL ANTISEPTICS.—Certain chemical substances are still largely used under special circumstances, and an exact knowledge of their mode of action is desirable, lest their employment should degenerate into a fetish.

The Factor of the Vital Resistance of the Living Tissues.—

AGE AND IDIOSYNCRASY.—Old people are much more liable to infection than young. This results generally from defects in the circulation. Certain individuals are much more liable to infection than others.

CIRCULATION.—Good general and local circulation is probably one of the most important factors in resistance to infection

Vascular parts, e.g , the head and neck, usually heal well in spite of infection.

Avascular parts, e.g , tendons, long skin flaps, are very liable to infection.

Morbid blocking of the vessels, e.g., in the feet of old people, makes ready healing without infection almost impossible.

Hyperæmia artificially produced promotes healing and enables the infected tissues to combat infection much more rapidly (*see* PASSIVE HYPERÆMIA)

Parts deprived of circulation are the most difficult to keep aseptic, hence the common sequence of suppuration upon gangrene.

EXCRETION.—

A healthy action of kidneys, skin, and lungs is most important.

In conditions of NEPHRITIS and renal insufficiency infection is common.

THE PROMOTION OF RAPID EXCRETION by copious infusion of saline solutions is one of the most potent agents in combating infection.

MORBID CONDITIONS OF THE BLOOD.—

DIABETES is the most notable condition in which the resistance to microbic invasion is much lowered.

NEPHRITIS, DEBILITY, AND MARASMUS act in the same way, though in a less degree

LOCAL CONDITIONS AFFECTING TISSUE VITALITY.—Any contusion of the wound predisposes to infection; hence the following often lead to suppuration:—

THE USE OF BLUNT INSTRUMENTS, lacerated and contused wounds.

Any lesion which causes BLOOD EXTRAVASATION, e.g., a hæmatoma.

PROLONGED HANDLING, dragging, cooling, or applying strong chemicals to the tissues.

Hence a NEAT AND QUICK OPERATOR who uses sharp knives and needles will get better healing than one who is slow and clumsy and uses blunt instruments.

Also multiple stitches which bring together tissues in layers without tension are less likely to suppurate than HEAVY SUTURES which grasp large masses of tissue with MUCH TENSION.

CONSTITUTIONAL IMMUNITY AND RESISTANCE TO INFECTION.—Apart from natural and individual immunity, over which we have no control, there are the following methods available by which the tissue resistance to infection can be increased:—

PROPHYLACTIC INJECTION OF ANTIBACTERIAL AND ANTITOXIC SERA, e.g., before an extensive operation for an ulcerating cancer of the mouth.

Curative Injection of Similar Sera after infection. The polyvalent antistreptococcus serum is of most use in this way.

Vaccine Treatment.—The injection of measured quantities of dead organisms grown from the source of infection greatly increases the power of resistance.

Injection of Material Causing Leucocytosis.—Nucleic acid and sodium cinnamate have been proposed, the former particularly when peritoneal infection is feared.

EFFECT OF CHEMICALS ON THE TISSUE VITALITY.—

Strong Antiseptics, e.g., 1–20 carbolic or 1–1000 perchloride, injure the living tissues, kill the leucocytes, and thus lower the natural vital resistance.

Weak Antiseptic Solutions, e g., 1–40 carbolic or 1–4000 mercury biniodide, injure the tissues very little if at all, whilst they decidedly inhibit the action of germs introduced from the air or skin.

Milton is probably the best of all antiseptics. It is 1 per cent electrolytic sodium hypochlorite containing 16·5 per cent by weight of sodium chloride, obtained from electrolytic cell.

The Character of the Tissues is of importance in this connexion. Serous surfaces have a great power of natural resistance to infection, but are readily injured by even dilute chemicals. Synovial surfaces, bone, and muscle tissue have less bacteria-resisting power, but are tolerant of weak antiseptics

The Exclusion of Bacteria from Wounds.—

BACTERIA IN THE SKIN.—These lie deep in the epidermis and in hair follicles and sweat glands, and cannot be destroyed with any degree of certainty. Hence, whatever skin preparation has been used, skin is always to be regarded as potentially infective and excluded from wound contact.

The Hands of the Operator and assistants are covered with sterilized rubber gloves and the arms by sterilized long-sleeve gowns.

The Skin of the Patient is clipped by its cut edges to sterilized towels as the first step of every operation

BACTERIA IN THE AIR.—Ordinary air contains only few bacteria. In still air the bacteria sink to the ground and are fixed by contact with any moist surface.

Air in Operating-theatre.—Is in some cases filtered through cotton-wool. In all cases dust is prevented from accumulating by providing non-absorptive surfaces with no crevices or sharp angles. All draughts have to be avoided, so that floating bacteria be not blown into the wound.

Air in Emergency Operating-rooms.—If there is ample time for preparation, a thorough cleansing and removal of all dust-carrying furniture and ornaments are desirable. But when there is less than twelve hours between the room preparation and operation, the less the dust is disturbed the better. The dust is much more dangerous in the air than on the carpet.

Air Expired by the Operator and Assistants.—Whilst quietly expired air has few microbes, that which accompanies speech is laden with the bacteria from the mouth. The operator and all who have to speak, leaning over the wound, must wear a **Mask** or respirator. A **Cap** prevents scurf falling from the hair into the wound, but a mask

The Exclusion of Bacteria from Wounds, *continued.*

serves the double purpose of excluding the bacteria from the head and the mouth.

GASTRO-INTESTINAL BACTERIA.—It is probably impossible to render the mouth or intestine sterile, but in healthy conditions the stomach and upper part of small gut are almost free from bacteria. Further, the number and virulence of germs in the mouth and large intestine can be greatly reduced by the following means:—

Mouth.—Removal of carious teeth, sordes, and tartar. Mouth-washes of astringents and antiseptics (e.g., alum gr x ad ℥j; protargol gr. j, glycerin ʒj, aq. ℥j).

Stomach.—Gastric lavage in diseases of or operation on the stomach. Feeding before and after the operation by sterilized food.

Intestine.—Aspiration of the upper part of the small intestine by means of a Ryle or other tube. A double-barrel tube allows of aspiration of the stomach and the giving of fluids at a different level. In cases of intestinal obstruction continuous intestinal suction is of great value

The Destruction of Bacteria.—

OUTSIDE THE BODY.—Here heat is almost universally employed, but antiseptics still have a useful place

Dry Heat has to reach 130° C. and be maintained for a very long time before the spores of bacteria are killed It is therefore seldom used

Steam Heat is used at a temperature of 100° to 120° C. for all dressings, coats, towels, etc. (*see* p. 84).

Chemicals are useful for materials which cannot be boiled, e.g., woven catheters; also for rapid sterilization, e.g., a towel required in an emergency operation wrung out of 1–20 carbolic, or a scalpel dipped in pure carbolic

IN THE TISSUES.—When bacteria are actually in the living tissues nothing but the living tissues can destroy them

ON THE SKIN.—When bacteria are on the surface of the skin a great deal can be done to destroy them, viz :—

Removal by Mechanical Means, e.g., scrubbing with soap and water

Fixation by such agents as alcohol, iodine solution, and rubber solution

Destruction by Antiseptics.—This involves a long and laborious process which is seldom entirely successful.

Preparation of the Patient's Skin.—

THE IODINE METHOD.—The parts are washed with soap and water and shaved if necessary This should be at least twelve hours previous to the iodine application, as the skin absorbs iodine much better when dry than macerated. The iodine is used as a 2 per cent solution in 80 per cent alcohol. Most commonly it is applied at the time of the operation. Some prefer to make two applications, one the day before and one at the time of the operation.

PICRIC ACID METHOD.—Picric acid as a 5 per cent solution in methylated spirit or a 3 per cent solution in water is an alternative to iodine, it has a longer endurance in the skin, and is free from the danger of forming a compound irritating to the eyes, such as arises from iodine and some kinds of spirit. Disadvantage that it stains linen badly.

'BONNEY'S BLUE.'—Contains 1 per cent of a mixture of equal parts of crystal violet and brilliant green dissolved in 50 per cent of rectified spirit and 50 per cent of water. The solution is painted on the skin prior to an operation.

Preparation of the Surgeon's Skin.—

THE SURFACES ROUND THE FINGER NAILS are exceptionally difficult to sterilize. Nails are to be kept short.

THE CONSTANT USE OF ANTISEPTICS will make the skin so cracked as to be impossible to clean.

FULL MECHANICAL CLEANING is impossible, as shaving and the removal of surface epithelium are its most important factor

THE HANDS OF THE SURGEON are liable to virulent septic contamination by contact with the mouth, rectum, or inflammatory products. Hence, whilst more difficult to sterilize, it is much more important for them to be sterile than the patient's skin

RELATION TO TIME.—Even if sterilization of the skin were possible, the time occupied makes such impracticable. For example; the passage of a catheter, which may take one minute, would have to be preceded by hand preparation taking fifteen!

USE OF GLOVES.—Hence rubber gloves are to be used as follows: (1) For every aseptic operation, to prevent infection by the surgeon's hands; (2) For every septic operation or manipulation, to prevent the surgeon's hands becoming infected by fresh and virulent organisms; (3) The hands are always cleansed mechanically and by spirit, or spirit and biniodide, before putting on the gloves, lest a puncture should occur.

USE OF SLEEVES.—The arms are covered by sterilized sleeves which fasten under the gloves.

Sterilization of Instruments.—

BOILING is the routine method.

TIME.—Twenty minutes is ample, even when the instruments have been infected. Five minutes is enough to kill all ordinary pyogenic organisms.

SOLUTION —Bicarbonate of soda ʒj to the pint is used. This prevents rusting, and the boiling-point is higher than plain water. The solution should be boiling at the time the instruments are placed in it.

PREPARATION.—All blood, etc, should be removed by scrubbing from the interstices before boiling, lest the germs should be protected by the coagulated albumin

SHARP INSTRUMENTS, especially scalpels, amputation knives, and razors, should be sterilized by ten minutes' immersion in 80 per cent spirit or pure Dettol. For rapid sterilization of any instrument in emergency, one minute in pure carbolic is much more efficient than hasty boiling

CATHETERS and other instruments made of woven material must be sterilized by immersion in 1–500 solution of biniodide of mercury, or by exposure to formalin vapour Some modern catheters are specially made to withstand boiling for a short period.

Sterilization of Ligatures.—

NON-ABSORBABLE LIGATURES, viz., wire, silkworm gut, silk, and thread, are boiled. But the last three are spoiled by repeated boiling.

CATGUT AND TENDON LIGATURES are largely used because they are absorbed in the tissues. The former is made from the submucous tissue of sheeps' intestines and needs careful preparation, especially in view of the fact that it may convey tetanus infection. Boiling in water destroys it.

Sterilization of Ligatures, *continued.*

CHEMICAL METHODS.—Saturation in sulphurous and then in chromic acid is Lister's method, which has never been proved faulty. Soaking in iodine is a more modern method.

THERMAL METHODS.—Boiling in xylol or other oils.

STORAGE.—Usually in alcohol or xylol, which preserves its hardness; or in 1–1000 watery biniodide, which renders it soft and elastic but difficult to tie firmly.

REGULATIONS RE MANUFACTURE OF CATGUT.—Catgut may now only be prepared for sale under licence from the Ministry of Health, under Therapeutic Substance Regulations, 1931. Individual preparation of catgut is discouraged and its sale prohibited. The Ministry allows the thermal method and the soaking in iodine method. Numerous tests for sterility are required. Importation of catgut is only permitted from firms subject to inspection; the Ministry has its Inspectors in U S.A., France, and Germany.

Sterilization of Dressings, Towels, Gowns, Gloves, and Swabs.—

BOILING is efficient, but not convenient, because the materials must then be used wet and cannot be transported.

ANTISEPTICS.—Soaking in 1–20 carbolic is efficient, and FOR TOWELS to be used for septic operations and in conditions where competent trained nurses are not available, is the safest method.

LOW-PRESSURE STEAM.—Fabrics are placed in an autoclave over boiling water. This raises the temperature to only 100° C., and the interior of large masses of non-conducting materials will not be raised to this unless the temperature is maintained for many hours. They are apt to be wet when taken out.

HIGH-PRESSURE STEAM.—This is the method almost universally employed.

ADVANTAGES.—By exposure to steam under high pressure a temperature of 110° to 120° C is reached, which is enough to kill the spores of anthrax or tetanus By exhausting the air from the sterilizing chamber before admitting the steam, the latter penetrates the interior of the largest masses of fabric By similarly exhausting the chamber after sterilizing the fabrics are dried.

DISADVANTAGES —A costly and complicated apparatus is required. Skilled, experienced, and trustworthy manipulators are essential for proper working. About two hours are occupied in the process. A colour index tube ought to be used with each packet of material. This changes colour at a given temperature, and shows that this temperature has been attained.

THE USE OF DRY DRESSINGS, quite apart from the method of sterilization, is a most important advantage. A wound will resist infection when dry which would suppurate if it were kept moist, because bacteria cannot grow in a dry environment

The Use of Special Antiseptics.—There are many chemicals, solutions, and powders recommended for special purposes. Only those of general use need be mentioned.

ALCOHOL.—Absolute alcohol is far too expensive. Ninety per cent alcohol in the form of 'surgical spirit' is a powerful antiseptic.

MERCURIC PERCHLORIDE OR MERCURIC BINIODIDE.—In strengths of 1–2000 dissolved in spirit or water are good antiseptics.

IODINE IN POTASSIUM IODIDE.—A 2–5 per cent solution of iodine in potassium iodide in spirit is a good general antiseptic.

IODOFORM is only an active antiseptic when it liberates nascent iodine in the tissues. The powder ought to be boiled, as in its dry state it may be contaminated by germs which it has no power of killing. Its chief use is in tuberculous abscesses and bone cavities.

FORMALIN or formaldehyde is used chiefly in sterilizing rooms or catheters, when it is employed as a vapour.

PEROXIDE OF HYDROGEN in solution is used for irrigation of suppurating and stinking cavities, e.g., an appendix abscess. The rapid liberation of nascent oxygen is its active principle, which mechanically cleans and stimulates the tissues and acts as a deodorant.

PROTARGOL (AND OTHER SILVER SOLUTIONS).—Generally employed in a strength of 1 to 4 gr. to the ounce. Is used for mucous surfaces—nose, mouth, urethra, or bladder. It acts both as an antiseptic and an astringent.

SODIUM OR POTASSIUM PERMANGANATE (Condy's fluid, 1–5000) is used for copious irrigation of mucous surfaces, e.g., the mouth and vagina.

MILTON is a useful all-round antiseptic. It can be used in every kind of wound, especially in septic wounds and burns. Milton is a 1 per cent electrolytic sodium hypochlorite solution containing 16·5 per cent by weight of sodium chloride.

THE CARREL-DAKIN METHOD.—The wound is excised carefully, and all grossly infected tissues and foreign bodies are removed. Small rubber tubes with lateral perforations are placed in all deep recesses. These are connected by glass and rubber tubes to a receptacle containing Dakin's solution. *Dakin's solution* is a fresh solution of hypochlorite, which must be of neutral reaction and contain 0·45 per cent of the drug. The solution is instilled into the wound at intervals of two to four hours. Outside dressings and tubes are changed daily, with aseptic precautions. The skin round the wound is smeared with sterile vaseline to prevent blistering. Twice weekly bacterial counts of the wound are made, and when infection has disappeared secondary suture is performed.

B.I P.P., i.e., bismuth-iodoform-paraffin paste, in proportions of 1, 2, and 3, is used for infected wounds. The grossly infected tissues are excised. The parts are dried and rubbed with spirit. B.I.P P. is then rubbed into all the wound recesses B.I P.P. gauze is packed in. The deep dressing need only be changed every two or three days.

CERTAIN ANILINE DYES are also used for infected wounds. The chief are brilliant-green and flavine

FLAVINE (1–1000 solution) is used after mechanical cleansing and excision.

MERCUROCHROME, 1·0 per cent or 0·5 per cent, is of particular use as a genito-urinary antiseptic.

SULPHONAMIDES have become the universal remedy for all pyogenic infections. The oral administration is more effective than the local.

PENICILLIN is giving excellent results with Gram-positive organisms in septic conditions. It is most useful in treating cases which have proved to be sulphonamide-resistant. Penicillin may well prove to be the most useful form of treatment for all septic conditions.

CHAPTER XI

SHOCK AND SYNCOPE

Definitions.—

SHOCK is the state of exhausted vitality resulting form and occurring immediately after injury.

SYNCOPE is a state of reflex inhibition of the cardiac and respiratory centres.

SHOCK

Varieties —

PRIMARY SHOCK —The immediate result of a severe trauma, especially of a blow on the head or abdomen Blood-pressure falls at once, which is probably due to a reflex cardio-inhibitory effect

SECONDARY SHOCK.—Develops slowly after a prolonged operation, and is usually the result of a combination of trauma, hæmorrhage, exposure, and toxæmia from the toxins of injured tissues.

Pathology.—

FALL OF BLOOD-PRESSURE is the cardinal fact, and its degree may be regarded as the measure of shock The systolic pressure, which is normally 110 to 120 mm., falls to 50 or 40 mm.

ARTERIES are greatly contracted

CAPILLARY STASIS —The capillaries are dilated, and the viscosity of the blood and the corpuscle content are increased in the capillary area.

VOLUME OF CIRCULATING FLUID is decreased owing to transudation as a result of increased permeability of capillary walls

VENOUS RETURN to heart is deficient.

DIASTOLIC FILLING is incomplete.

CORONARY CIRCULATION in inefficient

TOXINS FROM CRUSHED TISSUES —Cannon and Bayliss suggested that toxins as histamine entered the circulation from the injured area, and produced the effects of shock. Recently this work has been severely criticized by Blalock who has produced the effects of shock in uninjured animals by bleeding them so that 50 per cent of the blood volume was lost. Also transfusion of blood from a shocked animal failed to produce the effects of shock in an uninjured animal. O'Shaughnessy and Slome have produced similar evidence and also shown that shock only develops if the peripheral nerves are intact.

ACAPNIA, i.e , reduction of the normal CO_2 content of the blood. Excessive nerve stimuli cause excessive breathing, this reduces the CO_2. Further exposure of the intestines to the air causes rapid loss of CO_2. In the absence of CO_2 from the blood, the respiratory and possibly other vital nerve centres lose their normal stimulus; hence the activity of the respiratory, cardiac, and vasomotor centres is depressed.

Predisposing Causes.—

AGE.—

Infants under One Week display shock slightly, because the peripheral nervous mechanism is not fully functional.

Children suffer severe shock.

Old Age.—In old people the blood-pressure is higher than normal. There is a relatively small fall in the blood-pressure, but nevertheless the patient has feeble rallying power.

SEX.—Women are slightly less susceptible than men

NATIONALITY.—The highest races show most shock; Orientals and negroes comparatively less

TEMPERAMENT.—Nervous and highly cultured people suffer more than phlegmatic and dull ones.

TIME OF DAY.—Shock is most pronounced between midnight and dawn, especially with a sleepless night, i.e., a state of exhaustion. Least marked in the morning after a good night's sleep.

DEBILITY.—Cachectic states, especially that due to malignancy, anæmia following hæmorrhage; toxic conditions, especially those due to septic absorption: all conduce to severe shock

Exciting Causes.—

IN GENERAL TERMS, any peripheral or cerebral stimulus sufficient to exhaust the medullary nerve centres.

DEGREE OF SHOCK IS IN PROPORTION TO.—

Number of Stimuli, i e, of nerves stimulated or injured.—Hence injury of parts richly supplied with nerves, e g, the skin, testis, or hand, causes much shock.

Duration of Stimuli.—Hence the length of an operation is an important factor.

Intensity of Stimuli.—Rough injury, tearing, crushing tissues causes more shock than clean-cut wounds Skilful manipulation and use of sharp instruments give less intense stimulation to the tissues, and hence less shock

Proximity of Seat of Stimulation to Nerve Centres.—Shock is great in injuries of and operations on. The brain, spinal cord—Large cranial nerves, especially the optic and auditory—Large nerve plexuses, both sympathetic and spinal.

Presence of Contributory Factors which Lower Blood-pressure.—Of these hæmorrhage is the chief. Lowering of the body temperature and exposure of viscera act in a less direct manner. Conversely, the prevention of hæmorrhage, of cold, or exposure, and employment of means which directly maintain the blood-pressure, lessen shock

PSYCHICAL CAUSES —These acting alone usually produce syncope rather than shock. But when associated with injury they increase shock. Intense fear of an operation or horror in an accident may be the greatest factor in shock production

TOXIC AND CHEMICAL.—Usually these act slowly and cause gradual collapse. When they act suddenly they result in shock, e.g., the swallowing of corrosive poisons or the bursting of an abscess into the peritoneal cavity.

Shock—Exciting Causes, ***continued.***

THERMAL.—Burns often cause death from shock alone, and then one or more of the following factors are present:—

1 An Extensive Area of skin is affected—about one-third of the total skin area.

2. Age.—The patient is a child or a feeble old person.

3. Degree.—The burn is one of the 1st or 2nd degree, in which the nerve termini are irritated but not destroyed.

MECHANICAL.—

Traumatic Injuries, especially contused and lacerated wounds Clean-cut wounds and wounds by modern rifle bullets cause comparatively little shock in the absence of injury of important viscera.

All Surgical Operations.

Symptoms and Signs.—

BLOOD-PRESSURE.—This, as the cardinal factor, is regarded as the most direct measure of shock.

Normally the blood-pressure is 120 to 140 mm. of mercury.

Pathologically it is raised up to 200 mm. by old age, arteriosclerosis, granular kidneys, cerebral compression It is lower in various toxic conditions, anæmia, and marasmus.

Degree of Lowering.—It is commonly lowered below 100 mm. by severe operations Anything below 80 mm signifies danger; 20 mm. in the carotids represents the lowest point compatible with life.

CIRCULATION.—The arteries are contracted, and an undue proportion of the blood collects in the venous channels.

Pulse is short and quick and easily compressed, rising in severe shock to 140, above which it is difficult to count owing to the low blood-pressure

RESPIRATION.—The breathing is quick, sighing, and irregular.

Inspiration and expiration are shortened, and the pause between is lengthened.

FACE is blanched and shrunken, the nose pinched and dusky

The eyes are lustreless and rolled up beneath the lids

The eyeballs are deeply sunk in their sockets.

The jaw droops, the parted lips are pale.

SKIN is pale, cold, and clammy. Sweating is a marked feature and difficult of explanation. It may be due to the relaxed state of the skin or to toxic products circulating in the blood

SECRETION from other glands is diminished.

TEMPERATURE falls to 97° or 96° F. owing to the dilated condition of the cutaneous vessels and to the profuse sweating that occurs

NERVOUS SYSTEM.—

Cerebral Processes are diminished and the intellect wanders.

Muscles, both striped and unstriped, are relaxed and toneless.

Reflexes are diminished.

REACTION AFTER SHOCK.—Certain phenomena accompany the recovery from shock.

Blood-pressure rises slowly.

Pulse and Respiration become slower and more regular.

Vomiting occurs with the return of consciousness.

Consciousness returns after the reflexes have been restored.

The Temperature rises to about 99°.

Prevention of Shock.—

- GENERAL MEASURES.—
    - Rest, especially to the nervous system. A few days in bed before an operation, a good night's sleep, procured by an opiate if necessary, avoidance of alarming knowledge of the preparation for the operation.
    - Drugs.—Until recently stimulants, e.g., brandy or strychnine, were commonly given before an operation to prevent shock. According to the modern conception, however, a sedative is of more value. And therefore Morphia (gr. ¼ to ½) is given, and acts most powerfully by diminishing the sensibility of the nerve centres.
    - Maintenance of Temperature by warm clothing of the body and limbs, covering exposed viscera by hot compresses, and using a heated operating-table and a warm operation-room. It is essential, however, not to overheat the patients, as this further increases the shock.
    - Maintenance of Blood-pressure, especially in the head region. The head low, or Trendelenburg position. Firm bandaging of the limbs.
    - Avoidance of Hæmorrhage.
- THE ANÆSTHETIC.—The relation of shock to anæsthesia is a very complex one.
    - Full General Anæsthesia diminishes the capacity of the nerve centres for the receipt of stimuli. But such a degree of anæsthesia must in itself tend to lower the nerve vitality. Hence the general rule is to have full anæsthesia during skin incisions and certain visceral manipulations, e.g., in dilating the sphincter ani, dragging on adhesions, etc., and light anæsthesia for the rest of the operation.
    - Spinal Anæsthesia is the most potent preventive of shock. But it produces a fall of blood-pressure, which may be minimized by a preliminary injection of ephedrine It blocks the paths of afferent impulses during the operation, whilst avoiding the toxic effects of a general anæsthetic on the nerve centres. After the operation, when the action of the anæsthetic wears off, a modified degree of shock appears.
    - Nitrous-oxide-Oxygen anæsthesia has been proved by Crile to have many fewer shock-producing properties than ether or chloroform. As judged by the condition of the cerebral nerve-cells, it injures one-fourth as many as does ether in similar circumstances
- BLOCKING THE NERVE-TRUNKS.—In severe operations where the main nerves can be exposed and where spinal anæsthesia is not available, the injection of eucaine (gr. 2) or novocain (gr. 5) into the nerve-trunks is of great value. The chief examples of its use are in the forequarter amputation and in laryngeal operations, in the latter the superior laryngeal nerves are treated.

Treatment.—

- PREVENTIVE.—The above detailed measures should be carried out where applicable in order to prevent the further development of shock.
- STIMULANTS.—The value of all stimulants, e.g., alcohol, ether, digitalis, strychnine, is a matter on which great difference of opinion exists. All these remedies, especially alcohol (brandy ℥j in pint of hot saline per rectum) and strychnine (♏iij to v hypodermically), are still used, and common opinion supports their value. But when tested by the blood-pressure it is found that a short rise is followed by a lasting depression. They, accordingly, have been likened to 'whipping a dying horse' They

Shock—Treatment, ***continued.***

probably are of value in the less degrees of shock, and of doubtful utility in its more intense forms.

THE USE OF OXYGEN —When this is given it ought to be mixed with 10 per cent CO_2 in order to provide the necessary respiratory and vasomotor stimulus. Administered by a B.L.B. mask or nasal catheter.

BLOOD-PRESSURE RAISERS.—Opinion is still divided as to the value of pressor substances.

DESOXYCORTICOSTERONE ACETATE is of little value in the treatment of established shock, but it is said to prevent post-operative shock if administered in the pre-operative period.

PITUITARY GLAND EXTRACT is said to have a more lasting effect than the above

MECHANICAL AND THERMAL —Low head position, bandaging of the extremities, wrapping in blankets, and supplying hot-water bottles (*see* p. 89).

ADMINISTRATION OF FLUIDS —

In order to counterbalance the loss of blood volume, the circulating fluid must be increased. As a first-aid measure hot sweetened drinks are of value. In the shock-hæmorrhage syndrome whole blood should be administered. Plasma administered intravenously is of great value in the treatment of shock. Transfusion should be rapid at first, 1 pint in 15 minutes, and afterwards reduced to a drip It is important not to transfuse subjects suffering from blast injuries of the lungs, as the condition is made worse by it.

Recently it has been shown that injection of potassium phosphate solution (1 c.c. of $\frac{1}{8}$ gramme-molecule, pH 7·6) into the cisterna magna, has produced good results in the treatment of traumatic shock.

Fat embolism may produce symptoms resembling secondary shock, except that the blood-pressure is not markedly affected and is usually well maintained. As fat embolism can be made worse by transfusion, it is important to bear this condition in mind.

SYNCOPE

Syncope is the reflex inhibition of the cardiac and respiratory centres by psychical, cerebral, or traumatic causes

Causes.—

PSYCHICAL.—Emotion, especially fear or grief

CEREBRAL —Anæmia of the brain. Particularly when abruptly produced in anæmic persons, e g., by suddenly rising from recumbent position.

TRAUMA.—Sudden blows in the epigastric region Dragging upon viscera during operations. Dilatation of the sphincter ani. Operations in the region of the superior laryngeal nerve.

ATMOSPHERIC CONDITIONS, e.g , crowded hot rooms.

Relation to Anæsthesia.—Syncope during anæsthesia is usually due to the ANÆSTHESIA NOT BEING COMPLETE, allowing the nerve centres to be inhibited by the stimulus of the operation, e.g., by dilatation of the sphincter ani.

Also to cerebral anæmia in HEAD-UP POSITIONS—hence the danger of operations under anæsthesia in the sitting posture. If these must be undertaken, the patient should either be in a pneumatic suit, which allows of rapid driving of the blood to the brain, or else the position should be capable of alteration at a moment's notice.

Pathology.—Whilst syncope consists, just like shock, in a low blood-pressure with cardiac and respiratory inhibition, it differs essentially from it in its relation to the nerve centres. In syncope there is mere inhibition or anæmia, whereas in shock there is exhaustion. Hence syncope is easily and readily treated.

Symptoms and Signs.—Are practically identical with those of shock, but they are much more sudden in their onset, and hence during operations must be treated with greater promptitude. A tendency to VOMITING often occurs.

Treatment.—Head-down position. Rhythmic pressure of the heart. Artificial respiration Stimulants—alcohol, ether, or strychnine.

CHAPTER XII

ANÆSTHESIA

I. GENERAL ANÆSTHESIA

Choice of Anæsthetic.—This may be modified by: (1) The patient's condition; (2) The patient's age; (3) The nature of the operation.

From the numerous anæsthetic substances available, the general tendency is to select a combination of two or three and thus to obtain optimum anæsthesia, e g., avertin, gas and oxygen, and ether, or omnopon-scopolamine, evipan, and gas and oxygen.

Administration of Gas.—

PHYSICAL PROPERTIES —Nitrous oxide or laughing gas (N_2O). Stored in a liquid form in metal cylinders.

APPARATUS.—A 3-gallon rubber bag is filled from the gas cylinder and attached to a face-piece Valves are arranged so that air, gas, or a mixture can be given.

TECHNIQUE.—Patient is usually seated. Gag is inserted before application of face-piece. Few breaths of air are given. Pure gas is then breathed in and out of the bag The face-piece must fit accurately to exclude air.

SIGNS.—Breathing deep and stertorous. Pulse full and bounding. Clonic contractions, with jactitation of limbs Pupils dilate. Corneal reflex is lost

Time taken for induction, one minute

Duration, half to three-quarters of a minute

MODIFICATIONS —

PROLONGED GAS ADMINISTRATION may be induced for long operations, even those lasting for hours, by giving oxygen mixed with the gas in the proportion of 1 O_2 to 5 N_2O. From the point of view of ideal anæsthesia, absence of shock, and post-anæsthetic vomiting, this seems to be the best method.

The advantages are: The minimum of toxicity; the pleasantness of induction, the rapid recovery; the absence of shock or severe after-effects.

The disadvantages are: It is difficult to administer, there is often rigidity and struggling; it requires elaborate apparatus; it is very expensive. For major operations some basal pre-medication must be given, and ether may be necessary for full relaxation.

PHYSIOLOGY.—Nitrous oxide combines loosely with hæmoglobin. Exerts a specific action on nervous system. The effect on the circulation is stimulating.

AFTER-EFFECTS.—Practically nil. Headache, nausea, and vertigo occasionally.

Administration of Ethyl Chloride.—

PHYSICAL PROPERTIES.—Ethyl chloride (C_2H_5Cl). Very volatile colourless liquid. Highly inflammable. Stored in glass phials holding 50 to 60 c.c.

METHOD.—On an open mask, using 5 to 10 c.c., is the safest way. It can then be followed by ether if prolongation of anæsthesia is necessary. It may be given in a closed bag, but is then much more dangerous.

SIGNS.—Deep stertorous breathing. Dilated pupils, loss of corneal reflex. Relaxation of spasm of muscles. The masseter muscle is specially prone to spasm. Full pulse, with flushed face.

Time taken in induction, about 1 minute

Duration of anæsthesia, 1½ minutes.

AFTER-EFFECTS.—Headache and sickness in about a quarter of the cases.

Administration of Ether.—

PHYSICAL PROPERTIES.—Ether, $(C_2H_5)_2O$, is prepared from alcohol. Colourless volatile liquid. Specific gravity 0·72. Leaves no residue on evaporation. No reaction with litmus. Pure ether is prepared from rectified spirit. Pure methylated ether from methylated spirit. Both are equally good for anæsthetic purposes (Hewitt). *Special Danger*: Ether is highly inflammable, and its vapour admixed with air, and more so with oxygen, is exceedingly explosive. It should not be used when the diathermy, cautery, or any electrical instrument is to be used.

OPEN ETHER —Induce anæsthesia by chloroform, a mixture of chloroform and ether, or by a Clover's inhaler. In order to secure the best type of anæsthesia, administer three-quarters of an hour before operation a subcutaneous injection of atropine When anæsthesia has been established, place on the patient's face a gauze pad, or a pad of Gamgee tissue, with an opening for the nose and mouth, and over this place the mask covered with gauze or domette. Ether is now added to the mask to maintain the anæsthesia which has already been induced. Special care should be taken to maintain a free air-way.

ADVANTAGES.—Absence of cyanosis. Applicable to every type of case in which ether can be used. Safety.

DISADVANTAGES.—Low temperature of inhaled vapour. In some patients difficulty may be experienced in maintaining anæsthesia at the required depth. It is rather extravagant in the quantity of ether used.

THE CLOSED METHOD.—

A Clover's inhaler or one of its modifications

A closely-fitting pneumatic face-piece, connected with a rubber bag by a hollow shaft which passes through a metal chamber containing ether.

Adjustment is possible, so that the patient breathes air alone or varying mixtures of air and ether vapour.

Ether is only rarely used as a sole anæsthetic agent. It is therefore convenient to administer it through a Boyle or McKesson apparatus, as it can then be combined with gas and oxygen, or, if required, with chloroform.

SYMPTOMS.—

FIRST STAGE—ANALGESIA.—Swallowing, cough, holding the breath Pulse accelerated. Pupils large and mobile. Considerable degree of analgesia which will permit of simple surgical procedures.

Administration of Ether—Symptoms, ***continued.***

SECOND STAGE—LIGHT ANÆSTHESIA.—Unconsciousness. Loss of memory, intelligence, and volition. Struggling, shouting. Clonic contractions or tremor. Flushed face, with sweating. Pulse is quick and full Breathing often embarrassed by muscular spasm.

THIRD STAGE—FULL ANÆSTHESIA —Loss of corneal reflex. Muscular relaxation. Breathing regular and stertorous. Pupils moderately dilated; respond to light. Superficial vessels are dilated, and those of the head and neck especially so.

FOURTH STAGE—OVERDOSE.—Pupils widely dilated and immobile. Complexion of dusky pallor. Respiration shallow, irregular, or jerky. The circulation fails considerably later than the respiration, hence the great 'safety factor' with ether.

COMPLICATIONS.—

ASPHYXIA apart from overdose.

Is due to falling back of the tongue or jaw, spasm of the larynx or spasm of the respiratory muscles, inhalation of blood, mucus, or foreign bodies

Respiratory embarrassment is well marked, and the complexion becomes livid

Administration of Chloroform.—

PHYSICAL PROPERTIES —Chloroform ($CHCl_3$) is made from rectified or from methylated spirit, either variety being equally good for anæsthesia (Hewitt) Sp. gr. 1·49. Neutral to litmus No residue on evaporation. No colour when mixed with pure sulphuric acid No precipitate with silver nitrate.

APPARATUS.—

A towel or lint mask. A drop-bottle A Junker's apparatus (care necessary to see it is correctly assembled)

TECHNIQUE.—

PATIENT MUST BE LYING DOWN. Otherwise, when anæsthesia is induced, the blood is apt to gravitate to the lower part, and so by cerebral anæmia syncope is induced

Chloroform is put inside the mask at the rate of 20 to 30 drops, freely diluted with air, every thirty seconds until anæsthesia is attained.

Anæsthesia is maintained by giving the minimum quantity necessary every half minute.

THE CONDITION OF THE PATIENT is watched with minute care, especially as regards respiration, pulse, colour, and pupils, throughout the entire anæsthesia

SYMPTOMS AND SIGNS —

FIRST STAGE—ANALGESIA.—Restlessness, sighing, coughing. Flashes of light and noises in the ears. Sense of exhilaration and tingling. Some analgesia without unconsciousness.

SECOND STAGE—LIGHT ANÆSTHESIA.—Unconsciousness. Struggling. Some irrational talking. Flushing of the face. Pupils dilated and reacting to light

THIRD STAGE—FULL ANÆSTHESIA.—Breathing quiet and regular. Pupils contracted. They react sluggishly to light. The corneal reflex is absent. The eyes rotate downwards. The muscles are relaxed. The pulse remains normal in rate, but is more compressible

FOURTH STAGE—OVERDOSE.—This is the state of danger, and should never be induced purposely. The respiration becomes irregular or ceases. The pulse is feeble, irregular, or imperceptible. Pupils are widely dilated and do not react to light. Complexion is dusky or deadly pale. It is apt to occur suddenly from overdose at the beginning.

PHYSIOLOGY.—A momentary stimulation is followed by a lasting depression of the nerve centres. The highest cerebral centres are affected first. The lower centres in the medulla are affected later. When pushed to a fatal degree, paralysis of the respiratory centre occurs first and of the cardiac centre next. The 'safety factor' is low. Stimulants and arrangements for resuscitation should always be at hand.

THE PUPIL DURING CHLOROFORM ANÆSTHESIA.—

Dilatation occurs first from stimulation of the sympathetic. The pupil still reacts to light.

Contraction occurs later, when the sympathetic is paralysed, from stimulation of the oculomotor centre.

Dilatation occurs from paralysis of the oculomotor centre The pupil does not react to light.

DILATATION OF THE PUPIL occurs from five causes·—

Incomplete anæsthesia Vasomotor depression Vomiting	The pupil reacts to light.
Overdose of chloroform Asphyxia	The pupil does not react to light.

THE DILATED PUPIL WHICH DOES NOT REACT TO LIGHT is the most important and most urgent sign of grave danger. The pupil resembles the atropine pupil, being very widely dilated and quite immobile. It occurs in three conditions:

1. OVERDOSE, especially in chloroform anæsthesia.
2. ASPHYXIA of high degree, such as is seen occasionally when mouth gag has to be used throughout operation
3. REFLEXLY from an operative procedure, e.g., stretching the sphincter ani.

TREATMENT—Stop the anæsthetic and be prepared to begin artificial respiration at once, directly the signs of respiratory failure occur.

CESSATION OF RESPIRATION.—

CAUSES.—

Muscular spasm, with clenched jaw and fixed chest.

Tongue falling back so as to obstruct the upper aperture of the larynx.

Spasm of the glottis, especially in children.

Entry of blood, mucus, saliva, or foreign bodies into the larynx 5. Overdose of chloroform.	These two are very dangerous factors

Comparison of Chloroform and Ether.—

ADVANTAGES OF CHLOROFORM.—

1. FACILITY OF ADMINISTRATION.—No special apparatus is necessary. It is easy to give and pleasant to take.
2. COMPLETENESS OF MUSCULAR RELAXATION—This is specially noteworthy in abdominal operations, when the quiet shallow breathing and the relaxation of the parietes make manipulation easy

Comparison of Chloroform and Ether—Advantages of Chloroform, ***continued.***

3. ABSENCE OF IRRITATION OF THE RESPIRATORY ORGANS.—This gives it its value in children and old people who are liable to bronchitis, and in all who are suffering from disease of bronchi, lungs, or pleuræ.
4. ABSENCE OF VENOUS CONGESTION.—Hence its special use in cerebral surgery.
5. NON-INFLAMMABLE AND LESS VOLATILE THAN ETHER.

DISADVANTAGES OF CHLOROFORM.—

1. IT IS TEN TIMES MORE TOXIC THAN ETHER, and the death-rate from its use is ten times as high (1 in 1000 cases compared with 1 in 10,000).
2. ITS LETHAL EFFECTS ARE OFTEN PRODUCED SUDDENLY, with little or no warning, and may therefore occur with the most careful administration.
3. ITS ACTION AS A PROTOPLASMIC POISON, producing death by acid intoxication.

ADVANTAGES OF ETHER.—

1. THE SAFETY OF ITS ADMINISTRATION, being ten times less toxic than chloroform.
2. THE DANGERS HAVE WELL-MARKED SIGNS, consisting in obvious respiratory embarrassment, which cannot be mistaken by the most careless or inexperienced.
3. THE DANGER PERIOD IS OF COMPARATIVELY SLOW ONSET, giving ample time for remedial measures.

DISADVANTAGES OF ETHER.—

1. THE DIFFICULTIES OF ADMINISTRATION. It is unpleasant to take, and requires the knowledge of some apparatus.
2. IRRITATING EFFECTS ON THE RESPIRATORY ORGANS, with a tendency to production of bronchitis and pneumonia.
3. EXAGGERATED RESPIRATORY MOVEMENTS which embarrass thoracic and abdominal operations.
4. FREQUENT WANT OF COMPLETE MUSCULAR RELAXATION.
5. TENDENCY TO VENOUS CONGESTION, especially in the region of the head and neck.
6. RISK OF EXPLOSIONS.

Preparation of Patient for Anæsthetic.—

FLUID AND FOOD.—No food for six hours. A cup of tea, with glucose, within two hours may be allowed. Milk especially should be avoided, as it is not digested, and is then vomited in the form of hard curds. In cases where shock or hæmorrhage is anticipated, glucose solution given by the rectum is valuable.

BOWELS.—In all cases the rectum and large bowel should be emptied by an enema or aperient, but purgation in the sense of causing repeated watery stools is definitely bad.

BLADDER.—The bladder should always be emptied the last thing before the anæsthetic: if necessary, by catheter.

MOVING THE PATIENT.—The less the patient is moved after anæsthetizing the better. Hence the advantage of giving the anæsthetic on the operating-table. But with most patients the fear of the operating-room is more harmful than careful lifting. In lifting, the level supine position should be kept.

TEETH AND TIGHT CLOTHES.—Artificial or loose teeth to be removed, and any tight bands taken from the neck, chest, or abdomen.

STOMACH.—In cases of intestinal obstruction or in cases of emergency operations when no preparation has been made, the stomach should be washed out just before or in the early stage of anæsthesia.

DRUGS.—In severe abdominal operations MORPHIA (gr. $\frac{1}{4}$ to $\frac{1}{6}$) or OMNOPON (gr. $\frac{1}{3}$) lessens the amount of anæsthetic required, and also lessens the shock. SCOPOLAMINE, i.e., hyoscine hydrobromide, is often combined in $\frac{1}{150}$ gr. dose with $\frac{1}{75}$ gr. of atropine, and given before major operations, STRYCHNINE (liq. strych. ♏v) is given by some to counteract cardiac depression. CHLORETONE (gr. xv) is said to have a powerful effect in preventing post-anæsthetic vomiting in those who are known to be predisposed to this complication.

AVOID POSTURE PARALYSES.—

ERB'S PARALYSIS results when traction is exerted on the upper limb and the head turned to the opposite side. It is due to compression of the brachial plexus between the clavicle and 1st rib.

MUSCULOSPIRAL PARALYSIS is due to the arm being allowed to hang down over the side of the table, the table edge pressing against the inner and posterior surface of the humerus.

WARMTH.—The operating-room should be at least 70° F. Patient should be as warmly clad and as little exposed as possible. A flat rubber bag filled with hot water and wrapped in flannel should be placed on the table for the patient to lie on, in desperate cases.

Treatment of Emergencies of Anæsthesia.—

1 VOMITING DURING INDUCTION.—

Depress head well below level of trunk.

Of vital importance in cases of intestinal obstruction to avoid aspiration of vomit.

2. SYNCOPE OR CARDIAC FAILURE IN LIGHT ANÆSTHESIA.—

Depress the head lower than the trunk

Strychnine (♏x hypoderm.), brandy (℥j by rectum), ether (♏xv to xxx subcutaneously).

Artificial respiration.

If recovery takes place, the operation should not be resumed until full anæsthesia has been induced.

3. CARDIAC FAILURE IN FULL ANÆSTHESIA.—

Occurring suddenly early in chloroform anæsthesia the outlook is almost hopeless.

Occurring late in anæsthesia it should be recognized early enough to allow successful treatment.

Artificial respiration. Cardiac stimulants, e.g., coramine.

Direct heart massage through an epigastric incision.

4. RESPIRATORY EMBARRASSMENT WITH OBSTRUCTION.—

REMOVE THE OBSTRUCTION.—Push the jaw forward. Open the mouth. Pull forward the tongue. Clear the fauces and pharynx with sponge on holder.

LARYNGOTOMY OR TRACHEOTOMY for cases where the above fails to relieve obstruction, cases due to laryngeal spasm, and those due to pressure upon or foreign bodies in the larynx and trachea.

ARTIFICIAL RESPIRATION.—Stand behind patient's head. Grasp elbows and pull them upwards, then press them down and in on the chest. About 15 times a minute. It is useless to do artificial respiration unless obstruction is removed and the air-way kept clear.

Treatment of Emergencies of Anæsthesia, ***continued.***

Oxygen Administration combined with the above.

Venesection for stout plethoric persons in whom the asphyxia has caused marked venous engorgement, cyanosis, and cardiac embarrassment. Withdraw one pint of blood.

5. RESPIRATORY FAILURE apart from obstruction.—Rhythmical tongue traction. Artificial respiration.

The After-treatment of Anæsthesia.—

NECESSITY OF CAREFUL WATCHING.—Restlessness, with falling out of bed; hæmorrhage, external or internal; moving of splints, bandages, and dressings; asphyxia, from the pharynx becoming full of vomit: such chances require THE CONSTANT PRESENCE OF A NURSE until consciousness is regained.

POSITION.—

The Head is to be Kept Low until shock has passed off.

The Head to be Kept on One Side, to allow vomit to escape from the pharynx

WARMTH.—Blankets and hot bottles The latter must be outside the blankets and never touch the patient, otherwise burning may result.

STOMACH.—In cases of intestinal obstruction the stomach should be washed out before the patient leaves the table

FEEDING.—Slight cases require no special rule.

Withholding Food.—In severe cases no solid food is to be given for several days It is both useless and harmful, as the digestive processes are largely suspended

Giving Liquids.—The old plan of withholding fluids is to be condemned as cruel, harmful, and irrational. The patient wants water, and should be allowed to drink it freely, and in whatever quantity. If vomiting is persistent, water should be given by the rectum. In stomach and œsophagus operations only is there any need for restriction.

Nature of Food.—Plain milk and any food difficult of digestion are to be avoided. Peptonized milk, tea, meat extracts, barley-water, Benger's food must be given at discretion

Solid Food is usually given on the second or third day, after the bowels have acted

II. MODIFICATIONS OF GENERAL ANÆSTHESIA

Intratracheal Insufflation.—

ETHER.—

Method.—After preliminary general anæsthesia the larynx is cocainized and a flexible catheter (21 to 24 French gauge) or Magill's tube passed through the glottis until its end is 26 cm. from the teeth, i.e., at the bifurcation of the trachea A warmed mixture of air and ether vapour is pumped in by a continuous current at a positive pressure of about 10 mm. Hg. Respiratory exchange will then occur with very slight respiratory movements.

Advantages —It has all the advantages of a positive pressure apparatus, so that the chest can be fully opened without collapse of the lungs; it secures a free and unobstructed airway, so that cyanosis cannot occur; it overcomes the possibility of obstruction by the upper air-way; respiratory movements are diminished to those of the quietest sleep; it is of great value when the trachea is obstructed, e.g., in goitre cases;

it diminishes shock by relieving both respiration and circulation from the strain caused by usual anæsthesia; the constant stream of air escaping from the larynx prevents blood, mucus, or vomit from trickling down into the trachea.

DISADVANTAGES.—The difficulty of passing a catheter through the glottis and the possibility of damage—this is only a matter of experience; the danger of injury to the lungs or surgical emphysema by too high a pressure—this can be prevented by a safety valve in the apparatus which blows off at 15 to 20 mm. of mercury

Basal Anæsthetics.—

DEFINITION.—Substances used to reduce or abolish normal consciousness or induce forgetfulness, thus minimizing the fear of anæsthesia and operating theatres, and supplementing other anæsthetics.

WARNING —Most basal anæsthetic substances, if given in large doses, are dangerous because uncontrollable—i e, they cannot be withdrawn from the patient should untoward symptoms appear. They should never be given with the idea of producing anæsthesia—merely to prepare for it

SUBSTANCES GENERALLY USED.—

1. MORPHIA (gr. $\frac{1}{4}$) AND SCOPOLAMINE (gr. $\frac{1}{150}$) or OMNOPON (gr. $\frac{1}{3}$) AND SCOPOLAMINE (gr. $\frac{1}{180}$) —Excellent premedication before nitrous oxide and oxygen anæsthesia or before operations under local anæsthesia. After two or three hours the morphia may be repeated but not the scopolamine.
2 NEMBUTAL.—Oral administration in $1\frac{1}{2}$-gr. capsules; dose 1 to 3 capsules This produces drowsiness and allays fear.
3 AVERTIN (TRI-BROMETHANOL).—Administered per rectum in freshly prepared solution Avertin must be tested with special 'Congo red' indicator after being prepared before being administered to the patient The patient falls asleep in bed and does not wake for some hours after the operation
4 PARALDEHYDE IN OIL —Administered per rectum, dosage 1 drachm per stone of body-weight up to 8 drachms. This is a safe and simple basal anæsthetic, acting like avertin.

Individual anæsthetists will have their preference, depending to some extent on the type of operations performed and the facilities available.

Other Anæsthetics for Short Operations.—

EVIPAN SODIUM.—A powder which has to be freshly dissolved in distilled water—administered intravenously. It may be given in a continuous form with intravenous saline. Valuable for short operations which do not require much relaxation or only brief relaxation—e.g, dental extractions, setting fractures, removing small tumours, etc.

DISADVANTAGES.—Sometimes the patient remains drowsy for hours and cannot return home from surgery or hospital. Twitchings and jerkings may occur to hamper the operator.

PENTOTHAL SODIUM.—This is administered in the same manner as evipan and for the same operations. It is claimed to be safer and more reliable, and its effects are more prolonged.

DIVINYL ETHER.—A volatile liquid given by inhalation, using a bag. A powerful anæsthetic for brief operations and very suitable for children. Short recovery period, with few after-effects.

Anæsthetics for Short Operations, ***continued.***

CYCLOPROPANE AND TRICHLORETHYLENE are also used for short periods of anæsthesia.

WARNING.—These anæsthetic substances are safe within limits for reasonably healthy people. In the aged, debilitated, or dangerously ill, it is probably wiser to dispense with them and rely on more classical methods.

III. LOCAL ANÆSTHESIA

Drugs Used.—

COCAINE HCl.—Not used now as a hypodermic because of the danger of toxic effects. Used for ophthalmic work as a 5 per cent solution for instillation into the conjunctival sac. Used in nose and throat operations with adrenaline, because of its valuable effect in shrinking vascular mucous membranes.

CAUTION.—Should not be used in the urethra except in carefully balanced formulæ.

NOVOCAIN.—Dose up to gr v; gr. j is sufficient for small operations. Is practically non-toxic, and therefore should always be used for injection methods. Used in strengths from ½ to 2 per cent. This is the safe anæsthetic substance and has only two disadvantages: the patient remains conscious; and it may not be used in infected or potentially infected tissues. A vast variety of minor operations are performed under novocain alone and it finds an increasing use in supplementing anæsthesia in many major operations

COMBINATIONS WITH ADRENALINE.—An addition of 10 to 20 min. of adrenaline solution (1–1000) to the dose of either of the above causes marked vasoconstriction, and thus a limiting of the action of the solution to the part injected.

Adrenaline solutions cannot be boiled. They must be added after the other solution has been sterilized.

Methods and Their Application.—

INSTILLATION.—A few drops of 5 per cent cocaine solution placed on the conjunctiva render the latter and the cornea insensitive. Used for ophthalmic operations.

SPRAYING AND SWABBING.—Cocaine 5 to 10 per cent solution, usually with adrenaline, sprayed or painted on to the nasal, pharyngeal, or laryngeal mucous membrane.

NOSE.—For all intranasal operations which only involve the mucous membrane. The marked shrinkage of the swollen membrane makes the parts easily visible and reduces hæmorrhage.

THROAT.—As a preliminary to laryngoscopy and for small operations.

LARYNX.—For intralaryngeal operations, e.g., scraping tuberculous nodules or removal of papillomata.

SUBCUTANEOUS INJECTION.—Novocain should always be used, and cocaine definitely abandoned because of its dangerous toxicity. It ought to be used as an infiltration method, that is—

REGIONAL ANÆSTHESIA.—In this the drug is injected over a wide area, especially IN THE COURSE OF THE NERVE TRUNKS supplying the part. Examples:—

For Goitre.—Through two or three punctures the whole circumference of the goitre is infiltrated with a 1 per cent solution of novocain, without adrenaline. After waiting 10 minutes the skin incision is made, and the first stages of dissection are carried out. Then more of the solution is injected at the sides of the larynx and trachea.

For Hernia.—The solution is injected on the inner side of the iliac spine as well as over the hernia, so as to paralyse the sensory nerves (ilio-inguinal, etc.).

For Finger Operations.—An elastic ligature is placed round the finger at its base to further limit and intensify the action of the anæsthetic.

SPLANCHNIC ANÆSTHESIA.—This is used for operations on the upper abdomen, especially those on the stomach. The splanchnic nerves are given off from the lower thoracic sympathetic ganglia (great splanchnic 5 to 10, lesser splanchnic 10 and 11, lowest splanchnic 12) and pierce the crus of the diaphragm to join the cœliac ganglion. They are injected with 2 per cent novocain by a needle 12 cm. long thrust into the angle below the last rib, or through the abdominal incision from the front.

FREEZING BY ETHYL CHLORIDE SPRAY.—This is a barbarous method of opening superficial abscesses. It is extremely limited in value and should be abandoned.

IV. SPINAL ANÆSTHESIA

Although the scope of this method is not nearly so great as it promised to be on its introduction, it has undoubtedly a most valuable place among anæsthetics. It is impossible, however, yet to decide what is its mortality compared with general anæsthesia, because: (*a*) Many thousands of cases are necessary for comparison; (*b*) Spinal anæsthesia, in this country at any rate, is limited to cases of special gravity, whilst general anæsthesia is used for all trivial operations. Hence, what follows must be accepted with the limitations suggested.

Drugs Used.—

COCAINE HCl.—This was the drug first used, but is now abandoned because of its immediate danger and severe after-effects.

STOVAINE.—This used to be the drug most commonly used. Most people now prefer spinocain or percaine. It is much less toxic than cocaine.

Dose.—Gr. 1½, or 0·1 g., or 1 c.c. of 10 per cent solution.

Barker's Stovaine Solution.—0·05 g. stovaine, 0·05 g. glucose, to 1 c.c. water. The glucose is added to make a heavy solution which will remain in the lower part of the spinal theca.

Light Stovaine.—Made up with normal saline instead of glucose, so that it is of lower specific gravity than the cerebrospinal fluid. It is used for abdominal operations—e.g., excision of the rectum—where the patient is placed in the high pelvic position, in which position heavy stovaine would gravitate to the medulla.

SPINOCAIN (syn. Planocaine).—A solution of novocain and strychnine in a special solvent. It is of lighter specific gravity than the cerebrospinal fluid, and owing to its viscous character it does not diffuse, but floats like a bubble in the spinal fluid; it must therefore *never be given with the patient in the sitting posture.* The anæsthesia produced is controllable, and may be confined to the perineum or legs carried to the

Spinal Anæsthesia—Drugs Used, ***continued.***

umbilicus, or extended to the costal margin, according to the degree of the Trendelenburg position. Spinocain is practically non-toxic, and does not materially lower the blood-pressure. A disadvantage is that the anæsthesia lasts only from ¾ to 1 hour

PERCAINE (Howard Jones) —This is the latest spinal anæsthetic and is likely to displace most of the others Advantages are that duration of anæsthesia may last from 2 to 3 hours, and the drug is non-toxic and certain in action Large doses—from 8 to 18 c.c. of the solution—may have to be given, but much is done by judicious alteration of the level of injection. Segmental anæsthesia may be obtained

Method.—

For the puncture the patient lies on his side (for percaine the affected area should be uppermost), with the spine flexed as much as possible. The anæsthetist may afterwards control the level of anæsthesia by tilting the table

PREPARATION —Skin is cleansed over the seat of puncture.

EPHEDRINE —A preliminary injection of ephedrine will minimize the lowering of the blood-pressure

NEEDLE.—A needle 4 in. long, with a stylet and multiple openings, is necessary to prevent clogging by fat or blood-clot

PUNCTURE —Strictly in the mid-line of the back between the 3rd and 4th lumbar spines This is on a line joining the highest points of the iliac crests.

ENTERING THE THECA —The needle is manœuvred between the laminæ and then pushed on for about ½ in The stylet is withdrawn, and the spinal fluid ought to flow If it does not it is useless to inject the fluid.

PUNCTURE WOUND is closed with collodion

After-effects.—

Various unpleasant after-effects have been noted from time to time.

HEADACHE, which may be persistent and severe, occasionally occurs

Rarely · Various paralyses, even of oculomotor nerves, the bladder and rectal sphincters, or paraplegia, which usually soon recovers Persistent backache

Advantages of Spinal Anæsthesia.—

1. If the patient's general condition is good at the time of operation, then much less shock occurs at the time, and anti-shock treatment will then prevent secondary shock—e g , in excision of the rectum or amputation of the thigh for sarcoma. But if the patient is already shocked, then it is most dangerous—e g., in amputation of the thigh after a gunshot wound or injury.
2. It does not require special preparation of the stomach and bowels
3. In desperate operations likely to terminate fatally the patient does not die on the table and does not lose consciousness.
4. Very rarely a patient refuses to take a general anæsthetic, but will submit to a spinal injection
5. In certain conditions, e g , diabetes or acidosis, it is less dangerous than a prolonged general anæsthetic.

Disadvantages of Spinal Anæsthesia.—

1. IT FAILS IN SOME CASES.—In a varying proportion (5 to 10 per cent) the spinal theca cannot be penetrated satisfactorily, and in these the

anæsthesia is impossible. This is most likely in old, feeble, and emaciated patients.

2. THE PAIN OF THE PUNCTURE AND THE ALARM to nervous people, conscious of the operation.
3. THE RISK OF MENINGITIS OR LATE PARALYSIS.—This is very slight.
4. IN WAR SURGERY, it has been found that when profound shock already exists, the use of spinal anæsthesia is followed by a serious fall of blood-pressure which often proves fatal.

Indications for Use.—Operations below the umbilicus in which some of the following exist:—

1. Operations involving severe shock, e.g., amputation through the hip in debilitated subjects.
2. Diabetes, especially with acetonuria.
3. Certain conditions of heart weakness.
4. Desperate cases of intestinal obstruction with constant vomiting (This condition may be dealt with by the intratracheal method, p. 98.)
5. Operations performed *in extremis*.
6. When the patient refuses to take a general anæsthetic.

CHAPTER XIII

INJURIES OF BLOOD-VESSELS

HÆMORRHAGE

Definition.—Loss of blood from an external wound, or from a mucous surface, or into an internal cavity.

Epistaxis—bleeding from the nose

Hæmoptysis—from the lungs.

Hæmatemesis—from the stomach.

Melæna—blood passed by the rectum

Hæmaturia—blood passed with the urine

Symptoms.—

1 AFTER SUDDEN LARGE HÆMORRHAGE —

PALLOR of surface and of mucous membranes.

LIVIDITY exists if sudden death has followed wound of a large artery, because there has been no time for veins and capillaries to empty.

RESPIRATION quick, sighing, and gasping, the so-called 'air hunger'.

PULSE rapid, small, and weak.

NOISES heard in ears.

DIMNESS OF VISION with flashes of light

SYNCOPE, nausea, loss of consciousness

RESTLESSNESS

SURFACE IS COLD

SWEATING is abundant

SUBNORMAL TEMPERATURE.

GREAT THIRST.

2 AFTER RECURRENT SMALL HÆMORRHAGES.—

ŒDEMA of eyelids and extremities.

LIABILITY TO FAINT on slight exertion.

PULSE is quick and easily compressible, with full beat and empty artery between beats, and well-marked dicrotic wave.

A TENDENCY FOR ALL THE SYMPTOMS TO INCREASE as bleeding continues.

Blood shows LEUCOCYTOSIS and diminution of red cells and hæmoglobin.

After hæmorrhage has ceased, especially concealed hæmorrhage, TEMPERATURE rises to 101°–102° F. for one or two days.

Circumstances which Modify the Effects of Hæmorrhage.—

QUANTITY of blood lost.

RATE of loss.—The sudden loss of a comparatively small quantity is more dangerous than the gradual loss of a much larger quantity.

AGE.—Infants and aged patients bear loss of blood badly.

SEX.—Women are less affected than men.

CONSTITUTIONAL CONDITION.—The following render bleeding much more serious: Pre-existing Anæmia; any Asthenic or Cachectic Disease; MARASMUS; coexistent SHOCK; SEPSIS.

IN OPPOSITE CONDITIONS, notably in plethoric individuals with high-tension pulse, or any cause of visceral congestion, cerebral or pulmonary in particular, BLEEDING MAY BE POSITIVELY BENEFICIAL.

Diagnosis of Hæmorrhage (especially concealed hæmorrhage).—

IN SHOCK.—
- SYMPTOMS are at their worst immediately after the injury, and TEND TO IMPROVE with time, except in fatal cases.
  - NERVE SYMPTOMS are much more prominent from the first: Unconsciousness, flaccid limbs, pupils dilated.

IN CEREBRAL CONCUSSION.—
- Symptoms are of SUDDEN ONSET.
- UNCONSCIOUSNESS is well marked and out of all proportion to the circulatory depression.
- HISTORY of blow on the head

IN CEREBRAL COMPRESSION —
- PARALYSIS and coma.
- RESPIRATIONS are deep and noisy
- SKIN is hot and congested
- PUPILS are often unequal
- PULSE is slow and forcible

IN HÆMORRHAGE, on the other hand—
- SYMPTOMS are trivial at first and increase in severity gradually.
- Pallor, restlessness, dyspnœa, and thirst are conspicuous before unconsciousness occurs.
- (For the diagnosis of abdominal, thoracic, and joint hæmorrhage, see the respective sections)

The Arrest of Hæmorrhage always consists of two stages :—

1. TEMPORARY.—The stopping of the bleeding. This can be NATURAL OR ARTIFICIAL
2. PERMANENT.—The repair of wounded vessels This is always a vital process, and nothing artificial can take its place

Natural Temporary Arrest of Hæmorrhage.—

IS BROUGHT ABOUT BY:—
- THE CONTRACTION of the ends of the vessel.
- THE RETRACTION of the vessel in its sheath.
- CURLING UP of inner coats within the outer.
- COAGULATION of blood in and round the end of the vessel.
  - An internal clot forms between the nearest branch and the cut end of the vessel.
  - A central clot forms in the lips of the cut vessel.
  - An external clot forms outside the cut end of the vessel and between the middle coat and the sheath.

IS AIDED BY:—
- CONTUSION OR LACERATION of the vessel. This increases contraction and retraction of the coats.

Natural Temporary Arrest of Hæmorrhage, continued.

- *Diminished Blood-pressure Bleeding generally stops when the patient faints.*
- Increased Coagulability of the blood. This occurs in anæmia; in asphyxia; after taking much milk, lime salts, or magnesium carbonate; when fluids are restricted.

IS RETARDED BY:—

- Clean-cut wounds.
- Partial Division of vessels, preventing contraction and retraction of vessel.
- Increased Blood-pressure: Renal disease; plethora, inflammation and congestion, exercise
- Movement of the part.
- Stimulation of the Heart (by drugs, alcohol, etc).
- Hæmophilia.
- Jaundice.
- Diminished Coagulability of the Blood. Vegetarian diet, diminution of lime salts; citric acid; large quantities of fluid, restriction of food.

The Permanent Arrest of Hæmorrhage.—Produced by:—

HEALING OF THE WOUNDED VESSELS.—

- The clot becomes contracted and fissured
- The vasa vasorum dilate and exude plasma and white corpuscles
- The wall of the blood-vessel is thus swollen and more vascular.
- The clot is penetrated by leucocytes and fibroblasts, the latter formed by proliferation of endothelium
- The fissures in the clot are lined by proliferating endothelium, thus forming new capillaries.
- The capillaries communicate with (*a*) The lumen of the vessel; (*b*) The vasa vasorum
- The clot and the vessel wall eventually become organized into fibrous tissue.

Differences in Healing of Arteries and Veins —

- *In an Artery.*—A lateral wound does not heal until the lumen of the vessel is obliterated, because the high blood-pressure displaces the clot. When the lumen is once obliterated it is seldom opened up, because the thick arterial wall actively contracts and forms a strong scar.
- *In a Vein.*—A lateral wound, when once it is plugged by clot, heals. The blood-pressure is too low to disturb the clot. When a vein is obliterated by thrombosis, it often becomes re-canalized

The Surgical Arrest of Hæmorrhage is brought about by closing the mouths of wounded vessels and holding them closed until they are plugged by firm clot. The following methods are employed:—

1. PRESSURE.—

- As the Preliminary to other means: On the artery at a distance from the wound. On the bleeding point.
- As the Sole Means of arrest: Capillary or parenchymatous bleeding; bleeding from a superficial vein; bleeding in a bony cavity; bleeding from a hollow organ or cyst; deep hæmorrhage where ligature cannot be applied

Its Dangers in other cases are: Inefficiency; Gangrene; Conversion of open into concealed hæmorrhage.

2. FORCEPS.—As a preliminary to ligature; as a sole means in small vessels. Causes adhesion of lips of vessel and curling up of inner coats.

3. CLAMPS.—

Rapid Method.—A vascular pedicle is crushed and its vessels obliterated at one stroke, e.g., pedicles of polypi, appendix vermiformis.

Slow Method.—Clamps are left on for 48 hours. On inaccessible structures, e g., pedicles of renal tumours and broad ligament of uterus.

4 TORSION.—Vessel is twisted 8 half turns of the forceps Inner coats curl up inside lumen. Outer coats twist up as a knot outside.

5. LIGATURE.—The method of choice in most cases (*see* Ligature of an Artery in Continuity, p. 123)

6 DIATHERMY.—Either as a blunt cautery applied to a bleeding surface, or by touching the hæmostatic forceps applied to a bleeding point with the diathermy electrode

7. CAUTERY.—Causes great contraction and retraction of vessels. A dull red heat only is essential, otherwise the vessel will be clean cut.

Is Especially Useful · In parenchymatous bleeding; in bleeding from a sloughy surface.

8. COLD causes contraction of small vessels. Sucking ice for bleeding from mouth, throat, and stomach Iced douches for epistaxis Ice compress over a bleeding viscus, e g., in hæmatemesis.

9 HEAT acts in the same way as cold Hot water should be between 120° and 150° F Useful for large bleeding surfaces, e g., uterus, bone cavities, and cysts

10 STYPTICS, e.g., alum, perchloride of iron, and tannic acid They hasten coagulation of the blood. Very unreliable Small wounds of the face, capillary oozing in mouth or nose, fungating tumours.

11 SUPRARENAL EXTRACT as a local injection or application Injection up to 10 min of 1–1000 adrenaline chloride. Causes contraction of small vessels. Is generally used in conjunction with eucaine as a preliminary to superficial operations

12. ERGOT, administered by mouth or as a hypodermic injection Used for post-partum hæmorrhage. Causes the uterus to contract as well as its vessels.

13. HÆMOSTATIC SERUM—HÆMOPLASTIN.

Treatment of the Effects of Hæmorrhage.—

REST—local and general.

KEEP THE HEAD LOW—to prevent syncope

WARMTH—by blankets and hot bottles.

INFUSION OF SALINE FLUID (sodium chloride ʒj to Oj).—

Into a Vein.—Use 2–4 pints. Inject into a superficial vein, e.g., median basilic. Temperature 100° F. Avoid injecting air.

Subcutaneous.—Into the tissues under the mamma or axilla. Specially suitable for cases where assistance is unavailable.

Treatment of the Effects of Hæmorrhage—Saline Infusion, ***continued.***

CONTINUOUS INFUSION.—The saline solution is allowed to continue running into the subcutaneous tissue, or into the rectum, slowly for 12–24 hours, using 6–10 pints of fluid.

BLOOD TRANSFUSION.—The ideal method of treating anæmia.

CHOICE OF A DONOR.—Blood contains hæmolysins and agglutinins which may cause dangerous clotting if a suitable donor is not selected. All people fall into one of four groups as regards the properties of their blood. It is only necessary to test for agglutination, as this precedes hæmolysis.

TABLE I.—AGGLUTINATION

Group	Incidence	Serum agglutinates cells of group	Cells agglutinated by serum of group
1	3 per cent	0	2, 3 and 4
2	41 „ „	1 and 3	3 and 4
3	13 „ „	1 and 2	2 and 4
4	43 „ „	1, 2 and 3	0

To discover to which group a person belongs, test against serum of Groups 2 and 3 On one end of a slide place a drop of Group 2 serum, at the other end a drop of Group 3 serum. Add to these, with separate match-sticks, a drop of the blood to be tested, and stir well. If clumping takes place, in either or both, it does so within two or three minutes, giving a coffee-grounds appearance

TABLE II.—CLUMPING

Group	With Serum 2	With Serum 3
1	Yes	Yes
2	No	Yes
3	Yes	No
4	No	No

Group 4 is universal donor; Group 1 universal receiver.

For routine emergency work it is convenient to have several donors of robust physique belonging to Group 4 available, so that no preliminary testing will be necessary.

CITRATED BLOOD.—A litre bottle has 150 c.c. of isotonic sodium citrate solution (2 g. to 100 c.c. of distilled water) placed in it Blood is drawn from a distended vein by needle and cannula into the bottle, which is kept at blood-heat. Shake the bottle continually as the blood flows in, till a pint has been taken. The citrated blood is given intravenously to the recipient through a needle and cannula by pumping air into the bottle on the principle of the wash-bottle.

The advantages urged for this method are that clotting is impossible. Unfortunately, in practice, this often occurs in the rubber tube. It is claimed that the blood can safely be kept in stock till required. This may result in the degeneration of the corpuscles and the waste of donor's blood.

The Continuous Drip Method is most valuable, because it can be used whilst an operation is in progress. Either saline or citrated blood can be used, and the former can be injected either into a vein or the rectum. A reservoir with a capacity of 2 pints is fixed at a height of two or three feet above the patient. It is kept warm and the blood constantly mixed by having oxygen bubbled through it. In the tube proceeding from the reservoir is a glass chamber with a fine tube through which the fluid drips at the rate regulated.

RESULTS.—Blood-pressure readings after intravenous injections with saline, and fresh blood, show that saline raises the pressure for only two hours, when it falls down to, or even below, the original; while fresh blood effects an improved pressure for several days.

TRANSFUSION OF VASOCONSTRICTOR SOLUTIONS.—Adrenaline—Ergotinine—Pituitary gland extract. Raise blood-pressure, and therefor combat shock. Are unsuitable if the hæmorrhage is likely to recur.

RECTAL INJECTIONS —One pint of hot water, with or without brandy, given every two hours (also continuous irrigation, *see* p. 108).

HYPODERMIC STIMULANTS.—Strychnine hydrochloride up to ♏x of the liquor; ether ♏xx–xxx. These are indicated for syncope; they have the disadvantage of tending to renew the bleeding.

DIRECT STIMULATION of the heart by sinapism or fomentations.

DIET.—Hot fluid nourishment at first; light and generous diet later

DRUGS.—Iron, with quinine or nux vomica, for anæmia.

GENERAL.—Prolonged rest, with plenty of sun and fresh air

The Classification of Hæmorrhage.—

I. ARTERIAL: (1) PRIMARY; (2) INTERMEDIATE OR REACTIONARY; (3) SECONDARY

II. VENOUS.

III. CAPILLARY

I. ARTERIAL HÆMORRHAGE

OCCURS IN SPURTS synchronous with the heart's action.

Is bright red in colour.

Is much more profuse from the proximal than from the distal end of the vessel.

1. Primary Hæmorrhage.—

OCCURS at the time of the injury.

DUE TO a failure to occlude the wounded vessels

It tends to become less and cease in time.

TREATMENT.—

ENLARGE THE WOUND AND SECURE THE BLEEDING VESSEL.—Except:—

Wounds in the tonsillar region, when ligature of the external carotid is indicated

Stab wounds in palm and sole, where a graduated compress can be tried first.

TIE BOTH CUT ENDS in large vessels.

LET IT ALONE if the bleeding stops naturally, except:—

The distal end of a large artery.

A punctured wound of an artery.

Arterial Hæmorrhage, *continued.*

2. Intermediate or Reactionary Hæmorrhage.—

OCCURS within 24 hours of the injury.

DUE TO a failure of the means for the temporary arrest of hæmorrhage

BROUGHT ABOUT BY:—

Patient's movements and restlessness, or rise of blood-pressure, and increased force of the heart, which accompanies reaction after an operation.

The dislodgement of a clot from a vessel

The slipping of a ligature

Failure to secure the distal end of a wounded artery, which only bleeds after anastomosing vessels have dilated.

Failure to occlude the lumen of a punctured artery. The clot is displaced from the punctured wound when blood-pressure rises

TREATMENT —The same as for primary hæmorrhage

3. Secondary Hæmorrhage.—

OCCURS later than 24 hours after the injury, especially between 7 and 10 days.

DUE TO failure of repair in a wounded vessel, or ulceration of a vessel

CAUSES.—

a. SEPTIC INFLAMMATION of the artery

b YIELDING OF AN ARTERIAL CICATRIX, brought about by division of its inner and middle coats by too tight ligature; disease of the artery, e.g, atheroma or calcareous change; cachexia, hæmophilia, renal disease, diabetes: violent cardiac action; increased blood-pressure; punctured wound of artery, the lumen not being obliterated.

c. PROXIMITY OF LIGATURE TO A LARGE BRANCH, giving a very short cicatrix.

d. WOUNDS OF A BRANCH just below the ligature, whose cicatrix gives way when full force of anastomotic circulation is established

e FAULTY LIGATURE, which may be Septic—Too tight—Hard knot or sharp edge—Too quickly absorbed, e.g, catgut

PHENOMENA OF SECONDARY HÆMORRHAGE.—Is slight at first—Recurs at intervals—Becomes steadily greater at each recurrence—Has no tendency to spontaneous cure.

It is more frequent from the distal than proximal end of artery, because. at the distal side of a ligature there is smaller clot formed, the vasa vasorum are obliterated by ligature, the blood-pressure is higher.

TREATMENT OF SECONDARY HÆMORRHAGE —It should always be dealt with, whether it has ceased or not, and whether it is small or large.

a. PRESSURE ON MAIN VESSEL above should be tried first.

b. OPEN THE WOUND and ligature vessel above and below bleeding point. Endeavour to render the parts aseptic, and drain.

c. CAUTERY used at a dull red heat, in soft, sloughy wounds.

d. LIGATURE OF THE MAIN TRUNK above is indicated:—

When above methods have failed.

When it is known for certain what artery is bleeding.

After amputation at shoulder or hip-joint.

e. **Amputation** is called for when the bleeding occurs from the femoral artery already ligated in continuity, because a second ligature would cause gangrene. In the arm a second ligature may be attempted.

f. **Firm Plugging** is the only possible treatment in deep vessels of the root of the neck, groin, and abdomen.

II. VENOUS HÆMORRHAGE

OCCURS IN A STEADY STREAM, except:—

From the cerebral sinuses it spurts synchronously with pulse.

From large veins of the neck it spurts synchronously with respiration.

Is **of a Dark Colour.**

Is **from the Distal End** of the divided vessel only, except: in the neck, in varicose veins, in branches of large veins, if no valve intervenes.

TREATMENT.—

Pressure is sufficient in superficial veins.

Ligature is needed for veins in the neck—Deep veins—Varicose veins.

Plugging is necessary when veins lie in bony cavities, e.g., cranial sinuses.

Suture or **Ligature** in punctured wounds of large veins so as not to occlude the lumen.

Tie the Artery at a different place, but do not occlude the vein, if main vein of a limb is wounded slightly when operating to ligate the main artery in continuity.

III. CAPILLARY HÆMORRHAGE

(including Parenchymatous Hæmorrhage)

Consists of an oozing from wound surfaces.

TREATMENT.—Sew up the wound, or, when this is impracticable, pressure, plugging, or the cautery must be used.

HÆMOPHILIA

DEFINITION.—An hereditary disease in which there is a tendency to spontaneous hæmorrhages and very profuse and uncontrollable bleeding from wounds

INHERITANCE —The disease is peculiar to the male sex, but is transmitted by females, thus the sons develop hæmophilia, and the daughters who are free from the disease often beget hæmophilic sons.

PATHOLOGY.—Of this little is known Probably due to inadequate liberation of thrombokinase by the blood-platelets, which are abnormal qualitatively.

TRAUMATIC HÆMORRHAGES.—Except that the division of the umbilical cord is remarkably free from incident, every trivial wound is accompanied by steady and persistent oozing. Tooth extraction is often the first injury to call attention to the disease, and the steady bleeding from this may be fatal. Circumcision, or any other operation, will cause dangerous or fatal bleeding. The actual hæmorrhage is continued steadily as a capillary oozing for many days, and it is the consequent anæmia that causes death by syncope.

ECCHYMOSIS.—Slight or unnoticed trauma produces extensive subcutaneous ecchymosis; hence the sufferer is often called 'a bruiser and bleeder'. The bruises slowly disappear if no external wound exists.

Hæmophilia, *continued*.

JOINTS.—Bleeding into the joints is common, and produces changes of synovitis and osteo-arthritis eventually (*see* Chap. XXIII).

MUCOUS HÆMORRHAGES.—The mucous membranes are not so prone to bleeding as might be anticipated. But epistaxis, hæmatemesis, or melæna occasionally occurs in a severe or fatal form.

COURSE.—In bad cases the subjects seldom survive to maturity. About 60 per cent of the affected persons die before eight years. If they survive till after puberty the prognosis is better. But only 11 per cent survive to twenty-two.

TREATMENT.—From a surgical point of view it is emphatically negative It is most important to avoid performing operations on hæmophilic subjects, and where hæmophilic symptoms are present, to abstain from any active measures such as the cautery or aspiration of blood effusions. Prolonged rest and firm pressure are the only methods ordinarily available.

Both stitching and the cautery are useless, because bleeding occurs from the stitch-holes or from the cauterized surface.

The following methods have had some success:—

CALCIUM SALTS.—Calcium chloride in 30–60 gr. doses, or calcium lactate 10 gr. hypodermically in a 5 per cent solution.

BLOOD SERUM.—Fresh antidiphtheritic serum is a convenient form. It is given in doses of 20–30 c c. subcutaneously or 10–20 c.c. intravenously. It has been used as a prophylactic before necessary operations.

NORMAL BLOOD applied on a dressing to the wounded surface.

GELATIN, injected subcutaneously to increase coagulability.

ADRENALINE, ERGOTININE, injected locally to contract the bleeding vessels.

OVARIAN EXTRACT, given because women are so exempt from hæmophilia.

HÆMOPLASTIN (hæmostatic serum), 2 c c intramuscularly.

SNAKE VENOM (Russell viper) has been used with encouraging results.

OPEN WOUNDS OF BLOOD-VESSELS

These are dealt with under the subject of HÆMORRHAGE, and it only remains to note—

Incised Wounds bleed very freely, because there is but little injury to cause the inner coats to curl up.

Incised wounds which do not completely sever the vessel bleed most of all. because both retraction and contraction are prevented.

Lacerated Wounds bleed less than any, or not at all, because the inner coats are curled up by the violence of the injury, and the outer sheath is also twisted up as in torsion

Punctured Wounds—as when a bullet nicks one side of an artery—may result in a traumatic aneurysm; or, if the adjacent vein is wounded at the same time, an arteriovenous aneurysm, or aneurysmal varix, will result.

Treatment of wounded arteries and veins.—

LIGATURE both ends of divided vessel, or above and below a lateral wound.

Exceptions.—Large veins should have lateral wounds carefully sutured. Large arteries may also have lateral wounds sewn together with fine silk—but this is not the usual practice. The proximal end of a divided artery may be sewn to the distal end, so preserving the lumen.

SUBCUTANEOUS INJURIES OF BLOOD-VESSELS

Contusion and Laceration.—

CAUSES.—External blow, being run over, etc. Counter-pressure when reducing an old dislocation. Bullets of low velocity.

PREDISPOSING CONDITIONS.—Atheroma, or calcareous degeneration.

RESULTS—

Slight Injury to the Inner Coats, with proliferation of the intima and thrombosis.

Rupture of Inner Coats, with thrombosis, aneurysm from yielding of the outer coat, dissecting aneurysm.

Injury with Subsequent Sloughing of the Outer Coat, producing secondary hæmorrhage or aneurysm.

Injury to the Vessel may become Infected, causing septic arteritis, secondary hæmorrhage, or aneurysm

Rupture of all the Coats of the artery, with immediate thrombosis, or, more commonly, diffuse traumatic aneurysm.

Gangrene may follow a contusion of an artery: from simple thrombosis, especially if the artery is calcareous; rupture of the artery; diffuse aneurysm.

SYMPTOMS OF THROMBOSIS.—Loss of pulsation in the vessels beyond the injury. Possibly a tender swelling in the position of the injured vessel.

These may appear at once after the injury, indicating rupture of the inner coats; or may be delayed for several days, indicating thrombosis from contusion and proliferation of the intima.

SYMPTOMS OF ANEURYSM.—A pulsating swelling connected with the artery appears from a few days to a few months from the time of injury. Common mode of origin of axillary and popliteal aneurysms

SYMPTOMS OF RUPTURE, with the formation of a 'diffuse aneurysm'.—

Local Signs.—Sudden, sharp pain at the seat of injury. Patient may be conscious of a snap. Fusiform swelling rapidly appears at injured point. Pulsation and bruit sometimes present, often not.

Distal Signs.—Cessation of pulsation in vessels beyond. Limb is cold and numb. Lividity and œdema from venous obstruction.

General Signs.—Weak, rapid pulse. Pallor, dyspnœa, and syncope.

TERMINATION OF RUPTURE.—

1. Recovery.—The swelling becomes hard by coagulation. The ruptured ends of the vessel heal in the ordinary way. Collateral circulation is established.
2. Gangrene is produced by the pressure of the blood obliterating both the veins and collateral circulation when the main trunk is already severed.
3. External Rupture through the skin, and death from external hæmorrhage.

Contusion and Laceration—Termination of Rupture, ***continued.***

4 SUPPURATION of the blood mass, with subsequent bursting of the abscess, and death from secondary hæmorrhage.

5. DEATH from internal hæmorrhage. Especially if the ruptured vessel communicates with some internal cavity

TREATMENT OF SUBCUTANEOUS INJURIES OF ARTERIES.

1. IF THE SIGNS POINT TO THROMBOSIS:—
   Rest—Warmth—Aseptic preparation of the skin
2. IF THE SIGNS POINT TO RUPTURED ARTERY:—
   Put on a tourniquet above the injury if possible.
   Cut down on the swelling and turn out the clots.
   Tie both ends of the ruptured vessel. (It is possible to sew the proximal end of the vessel to the distal without obliterating the lumen This is not, however, often attempted.)
3. IF THE SIGNS POINT TO SUPPURATION:—
   If a tourniquet can be applied, proceed as in (2)
   If a tourniquet cannot be applied, tie the artery above the injury before opening the abscess.
4. AMPUTATION is necessary if gangrene or secondary hæmorrhage occurs

CHAPTER XIV

DISEASES OF ARTERIES

INFLAMMATION AND DEGENERATION OF ARTERIES

Varieties of Arteritis.—

ACUTE ARTERITIS.—

SIMPLE OR PLASTIC.—Producing the repair of wounded arteries.

SEPTIC.—Resulting from septic injuries and wounds.

EMBOLIC.—Resulting from the lodging of an infective embolus in the lumen of an artery.

CHRONIC ARTERITIS.—

ENDARTERITIS DEFORMANS —Atheroma. Chronic inflammation of the inner coats, of patchy distribution, affecting large vessels and resulting in dilatation.

ENDARTERITIS OBLITERANS (IDIOPATHIC, SYPHILITIC, TUBERCULOUS, OR DIABETIC).—Uniform inflammatory proliferation affecting the whole circumference of small vessels and resulting in occlusion

Simple, Plastic, or Traumatic Arteritis.—

PRODUCED by aseptic contusions and wounds.

RESULTS in proliferation of the tunica intima—Exudation from the vasa vasorum—Thrombosis of the vessel and conversion into a fibrous cord.

Is to be regarded as a conservative act of repair rather than disease.

Septic Arteritis.—

CAUSES.—A septic wound—A septic ligature—The extension of an abscess—Very virulent ulceration, e g , phagedæna (Infection from the blood-stream is considered under the next head.)

PATHOLOGY.—

The coats of the artery become acutely inflamed.

They are softened and disintegrated by the peptonizing action of bacteria and their toxins

The leucocytes and fibroblasts of the exudation are killed by the toxins, and form pus

The blood-clot in the vessel is either disintegrated by the same agents, or washed out by the blood-stream.

RESULTS.—Secondary hæmorrhage (*see* p 110) Hæmorrhage from phthisical cavities, from chronic ulcers, or from malignant growths, is generally the result of this process.

TREATMENT —That for secondary hæmorrhage, etc.

Embolic Arteritis.—Generally in young people. An infective embolus is dislodged from the aortic valves, mitral valve, or endocardium, in malignant endocarditis or acute rheumatism. It blocks a small artery.

Embolic Arteritis, *continued.*

RESULTS.—(1) Abscess, as in pyæmia, (2) Idiopathic aneurysm—an acute softening of the arterial wall yields to the blood pressure, and so gives rise to an aneurysm.

Atheroma.—

CONSISTS IN a chronic endarteritis, which results from long-continued strain, and produces degeneration of the arterial wall.

CAUSES.—Old age—Mechanical strain of laborious occupations—Chronically raised blood-pressure: e g., renal disease, gout, lead poisoning, alcoholism.

MORBID ANATOMY, ETC.—

DISTRIBUTION: Aorta—Large arteries—Coronary arteries—Arteries at the base of the brain—Splenic artery—Arteries of the leg more frequently than the arm—The curved arteries and the convex side of the curved portions are first affected—Points where large arteries bifurcate—Points where branches are given off from large vessels—Points where arteries curve round bones, e.g., the subclavian over the first rib—Points where arteries are subject to constant flexion, e.g , the popliteal.

NAKED-EYE APPEARANCE.—

VESSEL IS DILATED AND HYPERTROPHIED In some cases elongated and tortuous.

INTERIOR PRESENTS: Opaque oval white patches under the endothelium—Calcareous plaques bared of endothelium—Softened white swellings, the atheromatous abscesses—Shallow excavations into the tunica media, atheromatous ulcers

MICROSCOPICAL CHANGES —

Proliferation of the cells of the deep layer of the tunica intima.

This tissue is avascular, and soon degenerates: Fatty degeneration, or calcareous degeneration forming 'laminar calcification' of the artery.

The tunica media is invaded to some extent.

The endothelial lining, cut off from the vasa vasorum, is lost.

The tunica adventitia is thickened

RESULTS OF ATHEROMA.—

1. ANEURYSM—from yielding of the weakened wall.
2. DISSECTING ANEURYSM—by a forcing of the blood between layers of an artery at the edges of an 'atheromatous ulcer'.
3. THROMBOSIS—possibly gangrene.
4. RUPTURE from slight violence.
5. EMBOLIC OBLITERATION of the vessel by a detachment of a calcareous plate.
6. DEGENERATION OF STRUCTURES SUPPLIED, e.g., in the case of the heart (fibroid or fatty) or the brain (white softening).

TREATMENT.—Mainly medical except for complications. Light diet, with little meát and no alcohol. Gentle exercise. Keep the blood-pressure low by aperients or nitrites.

Idiopathic Endarteritis.—Produces changes similar to the last described. There is no evidence of syphilis. Coldness, numbness, and gangrene of the parts supplied. Great pain.

Syphilitic Endarteritis.—Occurs in the tertiary stage of the disease. Attacks small arteries. Brain, kidneys, and the region of gummata are most commonly affected.

CONSISTS IN: Proliferation of cells of tunica intima. The new layer of cells is vascularized from the vasa vasorum. Slight thickening of tunica media. Some thickening of adventitia.

RESULTS IN: Obliteration of the lumen of the vessel. Degeneration of the tissues supplied into gummatous material, or, in the brain, white softening with consequent paralysis.

TREATMENT.—Iodide of potassium.

Tuberculous Endarteritis.—Always occurs in the area invaded by tubercle. The small vessels are obliterated by a proliferation of the intima. Hence tuberculous areas undergo caseous degeneration.

(The ulceration which opens large vessels and causes serious hæmorrhage is always produced by secondary sepsis, e g., in the lung or intestine.)

Diabetic Endarteritis.—Similar to the above obliterating forms of endarteritis. Affects the anterior and posterior tibial arteries most commonly. Produces one form of diabetic gangrene

Degeneration of Arteries.—

FATTY DEGENERATION.—Affects the intima—Occurs in the aorta—Is difficult to distinguish from atheroma.

CALCAREOUS DEGENERATION.—Occurs in old people—Affects the tunica media—Muscle fibres are transformed into rings of calcified material—Occurs in medium-sized arteries, e g., brachial.

PRODUCES. Loss of arterial tone—Deficient circulation in the parts supplied—Coldness and numbness—Cramps, pain, and tingling—Thrombosis—Senile gangrene (the branches are calcified, and are therefore unable to dilate so as to form efficient collateral circulation)—Rupture from slight violence—Secondary hæmorrhage after operations

ANEURYSM

Definition.—A sac filled with blood or blood-clot communicating with the interior of an artery, the walls of which have become dilated.

Classification.—

I. IDIOPATHIC, or spontaneous.—(1) FUSIFORM, (2) SACCULATED, (3) DISSECTING.

II. TRAUMATIC.—(1) CIRCUMSCRIBED; (2) DIFFUSE, or ruptured artery.

III. ARTERIOVENOUS.—(1) ANEURYSMAL VARIX; (2) VARICOSE ANEURYSM.

IV. ANGIOMATA.—(1) CIRSOID ANEURYSM, (2) ANEURYSM BY ANASTOMOSIS.

I. IDIOPATHIC ANEURYSM

CAUSES.—

a. WEAKENING OF THE ARTERIAL WALL.—Atheroma—Syphilis (?)—Embolic arteritis—Partial laceration, or the cicatrix of an old injury.

b. INCREASE OF THE BLOOD-PRESSURE.—Violent exercise, especially when intermittent—Cardiac hypertrophy—Plethora—Chronic renal disease—Gout and lead poisoning.

Idiopathic Aneurysm—Causes, ***continued.***

c INDIRECT CAUSES, which bring about (*a*) or (*b*), or both.—

Age.—That of maximum activity—30–50 Aneurysms due to infective emboli are the only ones which occur more commonly in children.

Sex.—Males are ten times more liable than females, except in carotid aneurysm and dissecting aneurysm, which are commoner in women.

Occupation.—Soldiers, sailors, athletes, etc

Nationality.—Anglo-Saxons.

Strain.—Right arm is more prone than the left. Vessels which are liable to flexion and extension, e.g., the popliteal, are very liable to aneurysm

Alcoholism.

Symmetry, as seen, e g., in double popliteal aneurysm, is caused by identical conditions on the two sides

1. Fusiform Aneurysm

PATHOLOGY.—The weakened arterial wall yields in every direction to the blood-pressure (*Fig.* 25, B).

The vessel is thus elongated and dilated in its entire circumference

All three coats of the vessel form the aneurysm

The wall of the aneurysm is thicker than that of the normal artery

The inner coat is generally thickened by atheroma

The middle coat is thinned

The outer coat is thickened by fibrous tissue

Contains little or no clot.

DISTRIBUTION.—Aorta (common)—Largest arteries (rare)

COURSE —Of slow development and progress Comparatively symptomless. It may fill with clot, and be thus cured.

COMPLICATIONS.—One portion of the sac wall gives way, and a sacculated aneurysm is produced

TREATMENT —Only constitutional

2. Sacculated Aneurysm

PATHOLOGY.—One point in the arterial wall is weaker than the rest This may be a thin patch of atheroma, or a scar, or an 'atheromatous ulcer'. A local bulging takes place at this spot. The inner and middle coats disappear in this situation (*Fig.* 25, C).

THE OUTER COAT (added to by fibrous tissue) forms the only layer of the sac.

THE ABSENCE OF THE INNER AND MIDDLE COATS is of the utmost importance.

They form three-quarters to seven-eighths of the thickness of a normal artery.

They contain most of the elastic and muscular tissue to which the vessel owes its resiliency.

They contain the vasa vasorum, to which the vessel owes its nourishment.

THE SAC IS STRENGTHENED—

From the outside, by the addition of fibrous tissue and by the incorporation of surrounding structures

From the inside, by the deposition of laminated clot

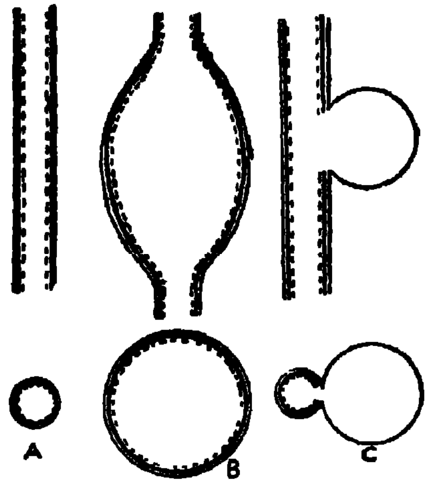

Fig. 25.—The essential changes of a fusiform and saccular aneurysm. Upper figures show vessel in longitudinal, lower in transverse section. A, Normal artery, showing three coats, B, Fusiform aneurysm, all the coats being equally stretched, C, Saccular aneurysm, in which the inner and middle coats have given way, the sac consisting of the outer coat only.

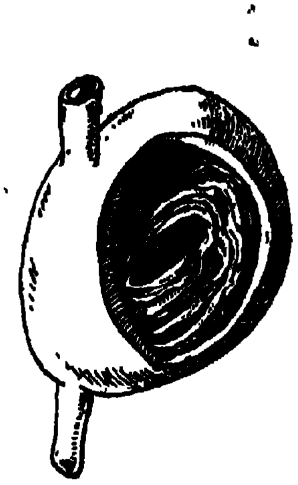

Fig. 26.—Saccular aneurysm, partly filled by laminated fibrin.

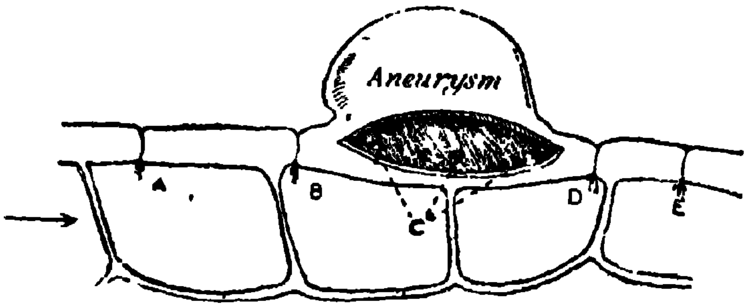

Fig. 27 —Diagram illustrating various ligations which may be used in the treatment of an aneurysm A, Distant proximal ligature—Hunter's operation, B, Anel's ligature, with no branch between ligature and sac; C, Internal orifices of vessel and branches sewn up in Matas' operation, D, Brasdor's ligature, E, Wardrop's ligature

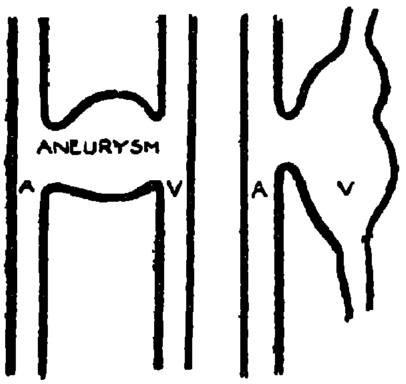

Fig. 28.—The left-hand figure shows diagramatically an arteriovenous aneurysm or varicose aneurysm, i.e., an aneurysmal dilatation of an artery which communicates with a vein. The right-hand figure shows an aneurysmal varix, i.e., an artery opening directly into a vein which becomes dilated owing to the arterial pressure. A, Artery; V Vein

Sacculated Aneurysm, *continued.*

CONTENTS.—

a. White, laminated, fibrinous clots at periphery (*Fig.* 26).
Generally in the parts least exposed to the blood-stream.
This is seldom organized into fibrous tissue, because of constant movement of pulsation, and absence of the tunica intima.

b. Red blood-clot of recent origin may be present ; always is when the aneurysm has lately been cured.

c. Fluid blood communicating with blood-stream.

RELATION OF THE CONTENTS TO THE SAC WALL.—The presence of laminated clot strengthens the sac. The presence of any clot lessens the fluid contents.

The pressure on the sac wall of an aneurysm depends on the area of fluid blood it contains, and varies as the square of the diameter of the fluid-containing cavity. It is thus a 'living Bramah press'

Effects of an Aneurysm.—

1. ON SURROUNDING STRUCTURES.—

EXCITES INFLAMMATORY REACTION.—Usually plastic. When infected it may be suppurative.

VEINS.—Compression, with cyanosis and œdema. Rarely a communicating perforation is formed.

NERVES are flattened out and destroyed. Great pain—rarely numbness and anæsthesia. Motor paralysis (rare, except in the case of the recurrent laryngeal).

BONES —Absorbed and eroded: notably vertebræ and sternum.

CARTILAGES are much more resistant than bones.

TENDONS AND FASCIÆ are incorporated in the sac.

MUCOUS CANALS are compressed and displaced.

2. ON THE CIRCULATION —

LOSS OF FORCE, i e., by blood-pressure beyond the aneurysm.
Partly compensated by cardiac hypertrophy and dilatation of anastomotic channels.

THE PULSE.—Is delayed. Is smaller and weaker. The impulse and dicrotic waves are absent.

AN ARTERY MAY BE BLOCKED by the cure of the aneurysm; by the pressure of the aneurysm on the vessel; by the involvement of a branch of the main vessel in the sac

Course and Terminations.—(1) CURE, (2) GROWTH AND RUPTURE; (3) SUPPURATION AND RUPTURE (or cure—vary rare).

CURE OF AN ANEURYSM occurs in three stages:—

1. FILLING OF THE SAC with blood-clot.
2. OBLITERATION OF THE LUMEN of the vessel between the aneurysm and the nearest collaterals. This is the essential condition of cure without which the final stage cannot occur.
3. ORGANIZATION OF THE BLOOD-CLOT into fibrous tissue.

SPONTANEOUS CURE may be caused by:—

Extension of the blood-clot from the aneurysm into the artery.

Pressure of the aneurysm upon the artery so as to obliterate it.

Displacement of laminar clot from the aneurysm into the artery as an embolus.

Arteritis excited by mere irritation (plastic), or by infection (suppurative).
Both of these are very rare.

RUPTURE OF AN ANEURYSM is much the commonest natural termination.—

THROUGH THE SKIN OR MUCOUS MEMBRANE.—

By small ulcer at first.

Bleeding like that of secondary hæmorrhage.

Externally, œsophagus, trachea, bronchus, stomach, or intestine.

THROUGH SEROUS OR SYNOVIAL MEMBRANE.—

By slit-like rupture.

Immediate large, generally fatal, hæmorrhage.

Pleura, pericardium, knee-joint.

SUBCUTANEOUS RUPTURE.—

Blood may extend without definite limit, producing gangrene as in a ruptured artery. Blood may be circumscribed for a time: the so-called 'leaking aneurysm'

SUPPURATION OF AN ANEURYSM.—Caused by infection of the tissues in and round the aneurysm

Their resistance may have been weakened by prolonged or rough pressure or manipulation, or sudden coagulation in a large sac.

Especially common in axillary aneurysm

Sac wall sloughs.

Artery above and below bleeds furiously when abscess opens.

Rarely the artery above and below is sealed by plastic arteritis and the aneurysm is thus cured

Signs of a Sacculated Aneurysm.—

TUMOUR.—Placed over and fixed to an artery Pulsates synchronously with the heart.

Pulsation is expansile, ceases when artery above is compressed.

A BRUIT is heard over the swelling, and sometimes a thrill is felt.

Systolic in time, blowing in character. Increased by pressure of the stethoscope. Rarely it is diastolic as well as systolic, especially in aortic aneurysms.

PULSE BEYOND is delayed in time and lessened in force.

PRESSURE SIGNS.—Congestion of veins with œdema. Paralysis of nerves. Muscular wasting.

PAIN.—Constant severe boring, especially when bones are eroded. Occasional lancinating pains in area of distribution of sensory nerves.

Diagnosis.—

ABNORMAL PULSATING VESSELS, e g., 'Pulsating Aorta'. In this there is no tumour.

TUMOUR OVER AN ARTERY.—In this the pulsation is heaving but not expansile; the pulsation ceases if the tumour is lifted off the artery; pressure on the artery does not affect the size of the tumour.

A bruit is sometimes heard, but is rasping and only occurs along the line of the vessel.

CYSTS OVER ARTERIES, which may communicate with joints.

Pulsation may be present and may be expansile. But pressure on the artery above does not lessen the size of the tumour.

If the cyst can be emptied into the joint, it fills out gradually and not by jerks.

Sacculated Aneurysm—Diagnosis, ***continued.***

PULSATING VASCULAR TUMOURS, e.g., Goitre and Sarcoma.

Tumour cannot be much reduced either by pressure or by compressing the artery.

Bruit is whiffing, and very variable in different parts.

The tumour can often be moved away from or along the main artery (e g., goitre).

The tumour is often in a situation where no large artery exists (e.g , the front of the head of the tibia)

Modification of the Signs of an Aneurysm.—

ANEURYSM UNDERGOING CURE —The sac is partly filled with clot. The pulsation loses its expansile character. Pulsation ceases when the lumen of the artery is obliterated

LEAKING OR RUPTURED ANEURYSM.—Rapid increase in size. Outline is indefinite. Pulsation and bruit are lessened or disappear. Pain and collapse Congestion, œdema, and numbness of parts below.

SUPPURATING ANEURYSM.—Increase in swelling, with indefinite outline Pulsation and bruit lessened Signs of acute inflammation. Œdema over tumour

Treatment of Aneurysm.—

CONSTITUTIONAL.—

Absolute rest, mental and physical

Diminished diet, especially as regards fluids, highly nutritious foods mainly of the nitrogenous type

Large doses of potassium iodide to reduce blood-pressure.

LOCAL.—

1. COMPRESSION of the artery above the aneurysm —
   - Digitally By relays of assistants acting 20 min at a time The thumb is reinforced by a 6-lb. weight.
   - Instruments, e g., the various tourniquets
   - Results of compression Often nothing, or
     - Aneurysm may be gradually cured by deposition of laminated clot
     - Aneurysm may be suddenly cured by clotting of all the blood in the aneurysm and vessel at once
     - The condition often recurs from a dissolution of the clot before organization has taken place.
   - Disadvantages of compression: Very painful, requiring anæsthetic or opiates. Digital requires so many assistants. Instrumental often causes sloughing of the skin. Is very tedious and uncertain. It causes enlargement of collaterals, and therefore subsequent cure by distant proximal ligature is rendered uncertain.
2. FLEXION OF ELBOW OR KNEE.—Only applicable to aneurysm at the elbow or ham when these are of small size.
3. ESMARCH'S BANDAGE.—Applied for 1½ hours under an anæsthetic. Followed by slight compression of the artery above for some days.
   - Is very uncertain. It may rupture the sac or cause thrombosis of the collaterals, with consequent gangrene.
     - (Methods (1), (2), and (3) are practically obsolete, and treatment is by surgical measures.)
4. LIGATURE OF THE MAIN ARTERY.—The routine method.
   - *a.* Proximal (*Fig.* 27): Close to the sac (Anel's operation); at a distance from the sac (Hunter's operation).

b. **Distal**: Used when proximal side of artery is out of reach. Main vessel (Brasdor's) (*Fig.* 27); main branches of the vessel (Wardrop's).

The above places of ligature are placed in order of efficiency (*see below*).

5 Excision of Aneurysm.—Preliminary tourniquet. All vessels connected with the sac are ligatures and cut. Sac is carefully dissected out.

6 Incision of Aneurysm (Matas' modification of the 'old operation').—Preliminary tourniquet. Sac is freely opened and emptied of blood and clot. The mouths of all vessels opening into the sac are sewn up by separate silk sutures (*Fig.* 27). The redundant portions of the sac are removed. The rest of the sac is folded over and sewn together as a solid pad under the skin

The use of stitches from the inner side is the distinctive feature of this operation.

Advantages over excision are—no risk of injury to veins, nerves, and collaterals which lie so close to the sac.

The only method available when the proximal ligature has failed

In addition to this, which is the obliterative method of Matas, there are two others, the conservative and the reconstructive The conservative method is for cases of sacculated aneurysm with narrow mouth communicating with the artery After opening the sac, the mouth of communication is sewn together. The reconstructive method is for a fusiform or a sacculated aneurysm with large mouth The lumen of the artery is reconstructed by sewing together the redundant walls of the sac over a channel leading from the afferent to the efferent artery The value of both these methods is doubtful, whilst that of the obliterative method is certain.

7. The Introduction of Foreign Bodies into the Sac.—Only used in inoperable situations, as in the aorta, and in the case of the abdominal aorta it has given recent successes. (*a*) Simple needles; (*b*) Needles connected with the positive poles of a battery; (*c*) Piano wire: many feet passed into the sac and left. May be passed at random through a cannula. Best passed by a special (Colt's) mechanism, as a complete spherical wire network

8 Amputation.—Ruptured or inflamed aneurysms (in certain cases). When gangrene or secondary hæmorrhage has resulted from ligature: Subclavian aneurysm (rare).

ON THE CHOICE OF THE METHOD OF TREATMENT —

Matas' Operation is the ideal treatment, and the only one when ligature has failed

Proximal Ligature close to the Sac is simple and efficient, but sometimes difficult because of the altered anatomical relations.

Proximal Ligature at a Distance from the Sac is easy, but liable to failure if the collateral circulation is too free, or to gangrene if it is not free enough.

Compression is to be used: In very nervous people who dread an operation; in patients with severe heart disease; in patients with extensive disease of the vessels.

The other methods are only suitable for special cases.

On the Ligature of an Artery in Continuity.—

LIGATURE MATERIALS.—

Silk.—Can be sterilized by boiling, but is unabsorbable, and therefore, if it become septic, may cause a sinus. It is the most generally used ligature.

Ligature of an Artery in Continuity—Materials, ***continued.***

CATGUT.—Requires complicated sterilization, and it is too quickly absorbed to be trusted for large arteries

KANGAROO TENDON AND OX AORTA.—Are absorbed more slowly than catgut.

METHOD OF TYING —

STAY KNOT should be used by taking two strands of ligature, tying the first turn of a knot in each separately, and then knotting the two pairs of ends together.

TIGHTLY ENOUGH to occlude the lumen without rupturing the inner coats of the vessel.

Ligature should be passed inside the sheath of the vessel. The vessel should be disturbed from its bed as little as possible.

LIGATURE OF MAIN VEIN.—The main vein should also be tied, because its ligature will cause a local rise in blood-pressure which helps to open up anastomotic blood channels

CHANGES PRODUCED IN THE ARTERY.—The lumen is occluded. The inner and middle coats are ruptured and curl up inside the vessel if the ligature is drawn too tight. Clot forms between the ligature and nearest branches above and below. Clot on the distal side of ligature is smaller and slower in formation than on the proximal. A portion of the occluded vessel is converted by the organization of the blood-clot into a fibrous cord.

ADVANTAGES OF APPLYING THE LIGATURE WITH MODERATE FORCE.—Inner and middle coats are not ruptured. Less liability to secondary hæmorrhage, especially when the vessel is diseased or when the ligature is very near a large branch Vessel walls are much thinner when distended by blood than after death. Outer coat is very thin, only one-twenty-fifth to one-quarter of the whole thickness.

LIGATURE FAILS TO OCCLUDE when: (1) Knot has slipped—In single-strand ligatures the beat of the artery opens the first turn of the knot whilst second is being tied. (2) Ligature is absorbed before organization of the clot has occurred.

EFFECTS ON THE CIRCULATION —The heart is embarrassed when a large artery is ligatured. Parts below the ligature become cold and pulseless; then the anastomotic channels dilate. Pulsation below the ligature is gradually resumed. Tissues become hyperæmic before they are natural. Some of the anastomotic channels permanently hypertrophy.

TREATMENT AFTER LIGATURE.—Parts should be kept warm and at rest in a horizontal position Any tight bandaging should be avoided.

GANGRENE MAY OCCUR AFTER LIGATURE.—

CAUSES.—(1) Circulation being too weak to open up anastomotic channels, either from a weak heart or as the result of a large hæmorrhage. (2) Diseased vessels unable to dilate for anastomosis. (3) Vessels contained in bony canals, e g , internal carotid and vertebral, cannot dilate. (4) Pressure of a tight bandage on anastomosing circulation. (5) Thrombosis extending down the vessels. (6) Cold or inflammation occurring directly after ligature.

EXTENDS.—Generally as dry gangrene of only one or two toes, or dry gangrene up to the ligature; or sometimes as moist gangrene up to the ligature.

Time.—Occurs third to tenth day after ligature.

Treatment.—Limited dry gangrene: wait to see the extent of natural repair. Extensive gangrene—dry or moist: amputate.

PROXIMAL LIGATURE CLOSE TO THE SAC (Anel's operation) causes obliteration of the artery and extension of the clot from the ligature to the sac.

It is most efficient because no branch intervenes between the ligature and the sac. It only causes one block in the circulation, and therefore only one set of anastomosing vessels is required. It is difficult to perform, because the aneurysm so distorts the anatomical relations.

PROXIMAL LIGATURE AT A DISTANCE FROM THE SAC (Hunter's operation) is easy to perform. The artery is more likely to be healthy. Causes a double block in the circulation: (1) At the ligature extending to the next branch; (2) Blood clots in the sac and extends to the vessel from which it springs.

Circulation has to be carried from the artery above the ligature by anastomotic channels to the artery between the ligature and the aneurysm. Thence by a second set of anastomosing vessels to the artery below the aneurysm. Hence two sets of anastomoses are required, and gangrene may arise by a failure of this double set.

Failure to cure occurs in Hunter's operation from too great freedom of anastomosing channels (this is often the case after long compression); the blood does not clot in the sac

Signs after Hunter's Operation when successful —Shrinking and loss of pulsation in sac Return of slight pulsation when the first anastomosing channels have dilated. Gradual consolidation of the sac as the second anastomosis is established.

3. Dissecting Aneurysm

AETIOLOGY.—A rare disease, commoner in women than men

ANATOMY.—Begins in the aorta and spreads down to the iliac arteries and up the carotids. The blood is forced between the coats of the vessels, separating the inner and middle from the outer It probably begins as an atheromatous ulcer. The folded inner coats block the lumen of the vessels.

SYMPTOMS.—Sudden, severe pain in the back and trunk, with coldness and pain in the legs from cessation of the circulation.

TREATMENT is impossible, and a fatal result occurs in a few days.

II. TRAUMATIC ANEURYSM

CAUSES.—(1) Contusion or subcutaneous rupture; (2) A small punctured or valvular wound of an artery; (3) The yielding of a cicatrix of a partially divided artery; (4) Closure of the external wound over a wounded artery.

DEGREES.—(1) Circumscribed, (2) Diffuse.

Circumscribed Traumatic Aneurysm.—The aneurysm is limited by a distinct sac. It is usually formed by the yielding of a cicatrix, or by the relaxation of pressure over a vessel some time after the injury. Its course is that of a spontaneous aneurysm.

OCCURS most commonly in the hand, foot, or scalp.

TREATMENT.—Pressure, excision, or ligature.

Traumatic Aneurysm, *continued.*

Diffuse Traumatic Aneurysm.—Occurs soon after the injury, and the blood collection is widely diffused in the fascial planes without a limiting sac

SIGNS.—A rapidly growing swelling in the line of an artery Pulsation and bruit are ill-marked. The skin is discoloured and tightly stretched The pulse is weak or lost below. Pain is intense.

RESULTS.—(1) External bursting; (2) Gangrene; (3) Suppuration

DIAGNOSIS has to be made from abscess or cellulitis by the thrill and bruit and great interference with the circulation. Also by its rapid development.

OCCURS most commonly in the axilla, groin, and ham

TREATMENT.—Incision and ligature above and below the wound

III. ARTERIOVENOUS ANEURYSM

DEFINITION —An abnormal communication berween any artery and vein (*Fig.* 28).

VARIETIES.—(1) Aneurysmal varix, (2) Varicose aneurysm

Aneurysmal Varix.—A dilated varicose vein communicating directly with an artery

CAUSES.—Stabs, phlebotomy, or gunshot wounds, which simultaneously wound a vein and artery. Very rarely idiopathic or congenital Most common at the elbow.

STRUCTURE.—The artery is enlarged and thickened, the vein is tortuous, and its branches varicose, with a large sac opposite the communication with the artery

SYMPTOMS AND SIGNS.—A fluid compressible swelling associated with varicose veins. Marked pulsation and a loud bruit are evident in the swelling, which partly empties on raising limb Pain may be severe, and congestive signs occur in skin below.

TREATMENT.—Pressure or ligature of the artery, or suture of the opening in the arterial wall

Varicose Aneurysm.—An aneurysm which communicates with a vein as well as with an artery.

CAUSES.—As above, and also by the pressure of an ordinary aneurysm opening into a vein.

SIGNS are the same as in the last, except that a more solid aneurysmal tumour may be detected in addition to the pulsating varicose veins.

TREATMENT.—Excision, with ligature of artery and vein.

(*Class IV is considered with other angiomata on p.* 132.)

CHAPTER XV

DISEASES OF VEINS AND LYMPHATICS

VENOUS THROMBOSIS

Definition.—A coagulation of the blood in the veins.

Causes.—

INJURIES OF THE VEIN WALLS —Contusion, laceration, compression, ligature.

INFLAMMATION OF THE VEIN.—Phlebitis All septic processes are apt to cause phlebitis and then thrombosis in neighbouring veins, e.g , appendicitis often causes femoral thrombosis

DEGENERATION OF THE VEIN —Varicose veins. The dilated cavities, rough coat, and sluggish stream frequently produce thrombosis

BLOOD CHANGES. DIMINISHED RATE OF FLOW

Varieties of Thrombi.—

RED THROMBI are formed by clotting *en masse*, so that the red cells are included

WHITE THROMBI consist of fibrin slowly formed, from which the red cells are absent

INFECTIVE THROMBI contain living micro-organisms

Effects of Thrombosis on the Vein.—

1. The clot may become organized into connective tissue and the channel permanently occluded.
2. The clot after becoming fibrous is calcified and forms a phlebolith.
3. The lumen of the vein may be re-established by gradual opening up of the interstices of the clot.
4. The clot may disintegrate before it has firmly formed If septic this produces pyæmia
5. The clot may be washed out *en masse* and form an embolus.

Effects on the Parts below the Thrombus.—

Great swelling and solid œdema (*see* PHLEBITIS, p. 129)

Enlargement of collateral veins

EMBOLISM

Definition.—A blocking of the blood-vessels by a substance carried thither by the blood-stream.

Regarded as to its effects, it is generally a disease of the arteries, but in its origin it is usually a disease of the veins.

Varieties of Emboli.—

SIMPLE.—

FROM THE HEART.—Vegetations from valves, or clots formed in dilated cavities

Embolism—Varieties of Emboli, ***continued.***

FROM THE ARTERIES.—Atheromatous plates, clots from an aneurysm.
FROM THE VEINS.—Thrombi.

AIR.—Introduced through a wound of a large vein, generally in the neck during inspiration. If large in amount it produces a frothy blood condition which clogs the heart and causes death.

FAT.—A not uncommon condition after bone injuries; the minute vessels of the brain and lungs are chiefly affected.

INFECTIVE.—Consist in infected portions of blood-clot and zoogloea masses of bacteria. Produce infarctions and pyæmic abscesses.

MALIGNANT.—Formed by the growth of soft, friable, malignant tissue, generally sarcoma, into the cavity of veins. Particles are washed away and cause metastatic growths, usually in lung.

PARASITIC.—Ova and scolices of tænia, filaria, etc.

Pathological Results of Embolism.—These depend upon: (1) The size and position of the vessel blocked; (2) The infective or simple character of the embolus; (3) The relation of the blocked vessel to the collateral circulation.

IN SIMPLE EMBOLI affecting terminal arteries:—

ANÆMIA.

NECROSIS (e g , white softening of the brain).

INFARCTION: a cone of tissue whose vessels are all thrombosed, the embolus being at the apex. The infarct subsequently organizes into fibrous tissue and cicatrizes.

HÆMORRHAGIC INFARCTION occurs in very vascular organs, e g., the lung, kidney, and spleen, and consists in a venous engorgement of the infarcted area.

IN INFECTIVE EMBOLI, infarction is followed by local suppuration forming *pyæmic abscesses.*

When the infected embolus lodges in an artery, an acute or inflammatory *aneurysm* may follow.

IN THE MAIN VESSELS OF THE LIMBS, gangrene may occur under conditions mentioned (*see* GANGRENE).

Special Clinical Forms of Embolism.—

CARDIAC AND PULMONARY EMBOLISM.—A large clot becomes entangled in the right side of the heart or the pulmonary artery Sudden death, accompanied by great pain over the heart, results.

IN THE BRAIN.—Generally the middle cerebral, supplying the motor cortex and motor tracts. Produces white or yellow softening and hemiplegia

IN THE RETINA.—Permanent blindness.

IN THE LUNG.—Sudden pain, dyspnœa, and hæmoptysis. Very rarely some local signs of pleurisy and consolidation. Simple infarcts cicatrize, infective ones form abscesses.

IN THE LIVER.—Usually infective, from the portal area, e.g., an appendicitis. Results in multiple abscesses.

IN THE SPLEEN OR KIDNEY.—Sudden pain in the affected organ, with occasional hæmaturia in the latter.

IN THE INTESTINE.—A large vessel is usually blocked. Tympanites, obstruction, and gangrene usually result.

Embolectomy.—Removal of an embolus from an artery. Has been successfully performed in main limb vessels. Rare successes have been reported in pulmonary embolectomy (Trendelenburg's operation). Operation must be immediate for pulmonary embolectomy and within a few hours in limb cases to give any chance of success.

METHOD.—Site of embolus is located, vessel exposed and lightly clamped on proximal side. Vessel is incised longitudinally and embolus removed. This is facilitated by releasing clamp, when arterial pressure will wash artery free. Clamp is again lightly applied, and vessel repaired with paraffined silk. During the post-operative period heparin should be administered to prevent intravascular clotting.

PHLEBITIS

Definition.—Inflammation of the vein wall.

Causes.—

SIMPLE.—Injury, pressure, gout, varicosity. Idiopathic cases are not uncommon.

INFLAMMATORY.—Thrombosis, spreading from a septic focus. Inflammatory lesions outside the vein. Septic wounds.

Pathology.—In simple cases a plastic organizing thrombosis occurs. In septic cases varying degrees of acute inflammation attack (1) the inner coats, (2) the whole vein, (3) the tissues outside the vein. In the last case an abscess is formed. Thrombosis usually spreads some distance up and down the vein.

Symptoms.—A hard, painful, cord-like swelling forms over the vein. Skin over this is dusky, congested, and œdematous. If the vein is superficial there are no other signs.

If it is the main deep vein of the limb, MASSIVE SOLID ŒDEMA occurs, with considerable lymphatic engorgement (white leg).

SUPERFICIAL VEINS ENLARGE in order to carry on the collateral circulation.

FEVER, with rigors, occurs, and is proportionate to the infectivity of the process.

ABSCESSES DEVELOP round an infective phlebitis.

Complications and Sequelæ.—

Cardiac or pulmonary embolism follows the dislodgement of a thrombus.

Pyæmia results from disintegration of a septic thrombus.

Permanent œdema, with varicose veins, is left in the leg when the deep femoral is blocked.

Treatment.—

Rest and elevation in bed for 6 weeks.

Belladonna application for pain.

Excision of the veins in recurrent superficial phlebitis.

Incision, removal of clot, and proximal ligature in infective phlebitis, e.g., in the jugular vein following acute mastoiditis.

VARICOSE VEINS

Definition.—A redundance, tortuosity, lengthening, and thickening of a group of veins.

Varicose Veins, ***continued.***

Distribution.—

Saphena veins of the legs, veins of the rectum (hæmorrhoids), and of the testes (varicocele).

Causes.—

Congenital over-development of the veins.

Absence or incompetence of the valves at the mouth of the saphenous veins.

Continuous over-distension caused by standing.

Increased abdominal pressure, from pregnancy, corpulency, or tumour.

Anatomy.—

THE INTERNAL SAPHENOUS VEIN is generally affected; the external more rarely.

The veins are dilated, thickened, lengthened, tortuous, and destitute of valves. A condition like atheroma frequently develops in their walls.

Localized pouches occur, or may constitute the main disease.

The tunica media is atrophied, the adventitia much thickened.

In extreme instances the vessel gapes when cut across.

Signs.—

The veins themselves form tortuous dilatations under the skin, chiefly on the inner side of the leg below the knee.

Congestion, œdema, eczema, and ulceration result from the impeded circulation in neglected cases This is most marked above the ankle and in front of the tibia.

Pain, tiredness, and aching are usual symptoms.

Complications.—

RUPTURE occurs from an injury of a thin pouch, or by ulceration. Bleeding may be severe.

PHLEBITIS is common and aggravates the congestive signs. Phleboliths may form in some of the veins.

ULCERATION in the lower half of the leg (as opposed to the syphilitic ulcer). Treated by injection of the veins and occlusive treatment with Elastoplast.

ECZEMA, or chronic infective dermatitis. Treated by injection of the veins.

PERIOSTITIS occurs in chronic ulcers

EMBOLISM is very rare.

Treatment.—

PALLIATIVE.—Superficial support by means of elastic bandages or stockings, adjusted before the patient gets up.

Exercise, as distinguished from standing, is beneficial, as promoting the deep muscular circulation.

Unna's paste is useful for all congestive and inflammatory conditions.

Congestive ulcers to be treated as already described (*see* p. 14).

RADICAL OR OPERATIVE.—This is required: (1) In candidates for military, police, and other public posts; (2) When congestive complications are present; and (3) When great pain and aching exist.

It is contra-indicated: (1) When the condition is secondary to deep thrombosis, coming on after, e.g., 'white leg'; (2) When it is only marked during pregnancy; (3) When it is diffuse, painless, and well relieved by elastic bandages.

OPERATIONS.—

TRENDELENBURG'S.—Consists in tying the saphenous vein just below the saphenous opening. It is indicated; (1) When a marked impulse occurs in the veins on coughing, showing a valvular incompetence; (2) When pressure over the top of the vein prevents the varicose veins from re-filling after they have been emptied; (3) When only the tributaries of the internal saphenous vein are involved.

SUBCUTANEOUS LIGATION OF THE VEINS WITH INJECTION is of great use, as with this treatment the patient is ambulatory.

INJECTION OF SCLEROSING FLUIDS —Injection of certain solutions into the vein lumen causes active thrombosis with formation of an adherent clot and final obliteration of the vein.

SOLUTIONS USED.—The solutions in common use are: sodium morrhuate, 10 per cent. Ethamolin (Glaxo Laboratories), 1–2 c.c., and sodium salicylate, 20–40 per cent, the latter is rarely used, as it is very irritating to the perivenous tissues, should any leak out into them. Quinine-urethane (quinine hydrochloride 4 g, urethane 2 g, water 30 c.c.)

METHOD.—Skin is sterilized Needle on syringe is inserted into vein, and blood withdrawn to be sure that needle is in vein; about 2 or 3 c.c. of sclerosing fluid are injected slowly. No anæsthetic is required. It does not involve the patient giving up work or remaining in bed. Further injections can be given at weekly intervals.

Moderate cases react well to injection, more severe cases require a combination of injection and Trendelenburg's operation. This can be done under a local anæsthetic.

NÆVI AND OTHER VASCULAR TUMOURS

Classification.—Tumours consisting entirely of blood-vessels are of four kinds: (1) Capillary nævi—birth-marks, port-wine stains, etc; (2) Venous or cavernous nævi—chiefly dilated veins, (3) Aneurysm by anastomosis—arteries and veins which open directly into one another; (4) Cirsoid aneurysm —purely arterial.

Capillary Nævi.—May be congenital, but usually develop soon after birth. Vary in size from a pin's head to half the body surface Usually bright red or dusky red colour. May be flat or raised and nodular. When very large (e g., half the body or over an entire limb) they are accompanied by hypertrophy of these parts When raised and nodular they often ulcerate and bleed

TREATMENT.—

THE APPLICATION OF SOLID CARBON DIOXIDE has now almost replaced the older methods of excision and electrolysis The snow formed by the sudden liberation of the liquid gas is moulded into the shape of a cylinder. The end of this is firmly pressed on the nævus, which it just covers, for about one minute. Vesication follows within a few hours, and a scab replaces this later. When this falls off the scar is thin and supple. No anæsthetic is required.

SURFACE APPLICATION OF RADIUM.—Short exposures on successive days often obliterate affected capillaries, leaving a barely observable scar.

Cavernous Nævi.—Usually affect the subcutaneous tissues as well as the skin. The veins are often large cavernous spaces into which arteries open

Cavernous Nævi, *continued.*

directly. A whole limb on one side of the body may be involved, and this is then generally hypertrophied.

TREATMENT.—This is only possible when the nævus is localized.

EXCISION is the ideal method for small tumours.

ELECTROLYSIS for the larger ones. A current of 200 milliamperes is passed through the positive pole in the form of a series of needles embedded in the mass.

RADIUM, applied as for capillary nævi. Treat by 'cross fire' irradiation if a tumour mass is present.

Aneurysm by Anastomosis.—A rare condition. It may be congenital, when it is associated with hypertrophy of the affected part. Masses of tortuous arteries and veins communicate freely with one another and produce a distinct bruit and thrill. It occurs in cancellous bones, or in the cranial diploe, or in one of the limbs.

TREATMENT.—The same as for cirsoid aneurysm.

Cirsoid Aneurysm.—A mass of dilated varicose arteries. Occurs most frequently on the scalp or orbit, connected with the temporal, posterior auricular, and occipital or ophthalmic arteries, but may arise on the perineum, trunk, or limbs. A loud bruit is heard over it The skin above and the bone beneath are atrophied by pressure. The hair falls out, and ulceration and hæmorrhage may result. Sometimes it spreads rapidly.

TREATMENT —Excision is usually out of the question. A series of ENCIRCLING LIGATURES or an attempt to ligature all the feeding vessels is the best method. Electrolysis is the only alternative

DISEASES OF THE LYMPHATICS

Acute Lymphangitis.—Occurs under the same conditions as cellulitis, into which it often merges. The inflamed lymph-vessels are seen as red and tender streaks running up to enlarged glands.

The treatment is that of the primary septic focus.

Chronic Lymphangitis may accompany syphilitic and tuberculous diseases.

Division of the Thoracic Duct, which opens into the junction of the left subclavian and jugular veins, is followed by a chylous fistula.

This must be treated by LIGATURE, anastomotic channels carrying on the chyle circulation

Lymphangiomata may be capillary or cavernous, and resemble nævi. They are often papillary, and the papillæ present clear vesicles. Their rupture gives rise to lymphorrhœa.

Cystic Hygromata, Macroglossia, and **Macrocheilia** consist of lymphatic dilatations of congenital origin (*see* Chaps. XXVII, XXIX, and XXXI).

Chylous Ascites and chylous hydrocele are very rare conditions due to obstruction to thoracic duct by tumours or parasites.

Chronic Lymphatic Obstruction is due to: (1) Tuberculous or malignant disease in the glands and vessels; or recurrent attacks of lymphangitis in cases of chronic ulcer or eczema. (2) *Filaria sanguinis hominis.*

SIGNS of this are: (1) Solid or lymphatic œdema; (2) Hyperplasia and connective-tissue overgrowth; and (3) Lymphatic fistula with lymphorrhœa.

ELEPHANTIASIS ARABUM.—Result of infection by *Filaria sanguinis hominis* introduced by mosquito bites. The adult worm is 3 inches long, and is located in a lymph- or blood-vessel; the swarm of embryos block the lymph-vessels and give rise to the obstruction. The external genitals and legs are chiefly affected, and form huge warty hypertrophied masses. Partial or complete AMPUTATION, or the ligation of the main arteries, are the alternative modes of treatment. Lymphangioplasty may be possible.

AFFECTIONS OF THE LYMPH-GLANDS

Acute Lymphadenitis is always secondary to some septic lesion in the associated skin areas.

ANATOMY.—The glands become swollen and matted together, and periadenitis quickly follows, producing a diffuse inflammatory swelling, which soon forms an abscess.

SITUATION.—

In the Submaxillary Triangle of the neck it is secondary to buccal, tonsillar, and dental diseases.

In the Posterior Triangular, to pediculosis or scalp ulcers or wounds.

In the Axilla, to sepsis in the arm or breast.

In the Groin, to sepsis of the genitals, abdominal wall, perineum, and anus (this generally affects the oblique set of glands parallel to Poupart's ligament); to sepsis of the leg, especially on the inner side.

In the Ham, to disease on the outer side of the leg and foot

TREATMENT is by fomentations and free incisions.

Chronic Lymphadenitis may be simple, syphilitic, or tuberculous.

Tuberculous Lymphadenitis.—

AETIOLOGY.—Children or young adults Bad food and hygiene. Frequently there is some predisposing cause for enlargement, e.g., carious teeth, tonsillitis, or pediculosis.

DISTRIBUTION.—The submaxillary, carotid, and supraclavicular glands are most often affected, and in this order; the axillary and inguinal glands much more rarely, owing to fewer predisposing causes for enlargement or points of entry for the tubercle bacillus.

PATHOLOGY.—The glands go through the following stages:—

1. Enlargement, due to a simple chronic hyperplasia of the lymphoid tissue; later, when tuberculous infection has occurred, development of typical tubercles in the lymphoid tissue ensues.
2. Caseation, sometimes followed by calcification or cicatrization.
3. Suppuration, which may be of a tuberculous or mixed type.
4. Periadenitis always occurs before long, and serves to mat together neighbouring masses of glands and to fix the glands to surrounding tissues.

SIGNS.—Three stages are usually noted:—

1. Simple enlargement, without much tenderness, in which individual glands can be distinguished.

Tuberculous Lymphadenitis—Stages and Signs, ***continued.***

2. The stage of periadenitis, in which the glands become fused together and more firmly fixed in their surroundings.
3. The stage of suppuration, when an abscess (usually cold) develops. Chronic sinuses are left by the bursting of the abscesses, and if these heal they leave scars which are puckered, keloidal, and vascular.

TREATMENT.—

PALLIATIVE.—High feeding, fresh air, and removal of all local sources of irritation.

ARTIFICIAL SUNLIGHT used in early stages.

OPERATIVE.—This is contra-indicated when active lung disease coexists. The glands must be dissected out or abscesses opened and scraped.

In the neck the incisions should be made (1) below and parallel to the jaw, (2) behind the sternomastoid, so as to leave inconspicuous scars. In bad cases it may be necessary to divide the sternomastoid or dissect out the internal jugular vein.

Operation should be followed by a period of sanatorium treatment.

DIAGNOSIS.—

SIMPLE CHRONIC ADENITIS affects only one or two glands, and is stationary.

SEPTIC ADENITIS has an acute course, accompanied by signs of inflammation.

HODGKIN'S DISEASE affects many group of glands simultaneously; the glands remain discrete longer, and do not break down Leucopenia is present

Other lymph-gland tumours (*see below*)

Other Varieties of Lymph-gland Enlargements.—

LYMPHADENOMA OR HODGKIN'S DISEASE.—Enlargement and overgrowth of lymphatic glands and lymphoid tissue of liver and spleen (*hard-bake spleen*) Glands remain discrete. There are three main types of the disease—cervical, mediastinal, and abdominal—according to the glands mainly affected Secondary deposits may develop in the lungs or bones, e g., the vertebral bodies.

TREATMENT.—Operation is useless. Arsenic and X rays

LEUKÆMIA.—Causes swelling of glands, bones, and spleen, with characteristic blood changes, i.e , great leucocytosis

LYMPHOSARCOMA.—Is a rare primary disease in the glands. It occurs in the neck and mediastinum. The growth is very rapid, vascular, and fatal.

SECONDARY MALIGNANT DISEASE.—Is common. Epithelioma or sarcoma may arise in the neck, axilla, or groin.

CHAPTER XVI

AFFECTIONS OF THE SKIN

Carbuncle.—A patch of infective gangrene affecting the subcutaneous tissues.

CAUSES.—Infection with *Staphylococcus pyogenes aureus*. Albuminuria or diabetes are common antecedents Local abrasions or contusions sometimes precede.

PATHOLOGY.—Acute inflammatory exudation is caused by the cocci. The dense fibrous tissue under the skin does not yield, and the compression strangles the vessels. Septic gangrene results from these causes. Later, the living tissues suppurate round the septic slough and form pus and granulations.

Hence, the following zones are distinguished from within outwards: (1) Central slough or core, (2) Layer of pus; (3) Layer of granulations; (4) Inflamed tissues.

Then the pus breaks through the overlying skin in one or two places and the slough is discharged, the cavity being filled up by granulation and cicatrization.

SIGNS.—Localized, red, brawny, painful, swollen area of skin, from one to six inches in diameter. There may be slight enlargement of lymph-glands. In one or two weeks the skin becomes vesiculated, and then shows several grey points. These burst, and pus oozes out. The openings run together, and some time later the slough comes away, the suppuration becoming freer all the time. The cavity left heals by granulation.

Extension may occur, especially if septic poultices are used, by infection of neighbouring hair follicles.

DISTRIBUTION.—Nape of neck, back, nates, and face are the commonest sites. Usually single.

COMPLICATIONS.—Septic thrombosis of the sinuses may occur with carbuncle of the face. Septicæmia, pyæmia, and acid intoxication with coma—especially in diabetes.

SYMPTOMS.—General malaise, without much rise of temperature. If general septic infection occurs it will cause the usual symptoms.

DIAGNOSIS.—

BOILS are multiple, conical, and have only one opening.

GUMMATA are painless, with but little inflammation. The edges are clean cut and sharply defined, the discharge is gummy, the slough like wet wash-leather.

ANTHRAX.—A black slough surrounded by a ring of vesicles.

Very marked œdema and swelling of lymph-glands. Little local pain, but marked rise in temperature.

PROGNOSIS is good, unless albuminuria or diabetes exists, or sinus thrombosis or general septic infection occurs.

Carbuncle, *continued.*

TREATMENT —

EXPECTANT.—Hot fomentations followed by a dressing of magnesium sulphate and glycerin when the discharge commences. Short-wave diathermy.

OPERATIVE.—A crucial incision with a thermo- or diathermy-cautery and removal of dead tissue by the same means.

CHEMOTHERAPY.—Administration of sulphathiazole.

A Furuncle or Boil is essentially the same as a carbuncle. A boil differs from a carbuncle in the following: It affects the skin only, and not the subcutaneous tissue. It opens by a single opening, instead of several. It is multiple very frequently. Usually occurs from the infection of a hair follicle or sweat gland. FURUNCULOSIS is a disposition to repeated crops of boils.

TREATMENT.—

LOCAL AND PROPHYLACTIC.—Lotions of spirit and biniodide, 1–1000.

GENERAL.—Injection of staphylococcal vaccine prepared from the organisms actually present in the boils. Improve general health, injections of collosol manganese Penicillin.

Corn.—A hard keratinous mass of epidermis with a deep-growing conical centre, which presses on the tender parts of the deep skin.

CAUSES.—Abnormal pressure—badly fitting boots, projecting bony processes.

LOCALITY.—Toes, especially little and great, and over the prominences of talipes.

VARIETIES.—Hard or soft, according to whether they are exposed to the moisture of sweat or not.

TREATMENT.—Excision. Salicylic acid ointment or lotions Pad to relieve pressure.

Wart.—Papilloma of the skin. Often multiple, and then usually on the hands of children. They appear sometimes quickly in crops, and disappear equally quickly. Possibly they are locally infective, i.e., they disseminate themselves.

TREATMENT.—Soften with salicylic ointment, and then touch with pure acetic or nitric acid.

PLANTAR WART.—Usually single on the plantar surface of the great toe.

TREATMENT.—Excision, taking care not to penetrate the dermis

VENEREAL WARTS are very large villous masses, which occur on the external genitals, glans, prepuce, or labia, as the result of the irritation of discharges.

TREATMENT.—Excision, with cautery of the base; or application of calomel or acid nitrate of mercury. Essential to keep the affected area clean and dry

Lupus Vulgaris.—A very chronic tuberculous disease of the skin.

OCCURS in children, or adults under 30. Attacks the nose, cheeks, lips, eyelids, nasal, oral, and lachrymal mucous membranes, and ears. Less often the fingers, toes, or trunk.

SIGNS.—Nodules appear deep in the skin, and then run together. Each nodule is red and raised, and resembles 'apple jelly'. The skin becomes infiltrated and ulcerated, fresh nodules appearing on the spreading margin. One edge may heal whilst the other extends. The ulcer is covered with granulations, and over these are thick scabs formed by the drying of the purulent secretion. Deep tissues, e.g., the cartilages of the nose or ears, are attacked and destroyed. The scars left are puckered, thin, and vascular.

COURSE.—The disease runs a course of many years, but occasionally dies out spontaneously.

COMPLICATIONS.—Epithelioma may form in the scar. Other tuberculous diseases very rarely arise.

DIAGNOSIS depends on the tuberculous nodules, the slow superficial ulceration, the congested scar tissue.

SYPHILITIC ULCERATION is much more rapid and deep. Other signs are usually present. The scars are thin, pale, and supple.

LUPUS ERYTHEMATOSUS.—*See below.*

TREATMENT.—Removal by scraping, and the subsequent application of caustics, e.g., zinc chloride. Improve the general health of the patient. Finsen light or X rays.

Lupus Erythematosus.—

OCCURS generally in adult women, on the face in symmetrical patches, like a butterfly, the wings on the cheeks and the body on the nose.

SIGNS.—Smooth hyperæmic patch, covered with a fine branny desquamation. Associated with some seborrhœa. It does not ulcerate, but leaves a thin white scar surface in the middle, whilst the edge spreads.

TREATMENT.—Tar and mercury ointments Finsen light

AFFECTIONS OF THE NAILS

Onychia, or Perionychia.—An inflammation of the nail matrix.

SEPTIC ONYCHIA is an ungual whitlow caused by a septic wound. Pus collects under the nail

TREATMENT.—Removal of the nail and scraping the granulations.

SYPHILITIC ONYCHIA occurs in congenital and acquired syphilis in the secondary stage. It yields to specific treatment.

Ingrowing Toe-nail.—Usually affects the great toe

Caused by boot pressure, the edge of the nail being pressed into the soft tissues and causing a chronic ulceration.

TREATMENT —Removal of nail in severe cases; in others local wedge resection of the overlying skin and nail-bed.

Onychogryphosis.—A deformed and overgrown great-toe-nail seen in old people When it becomes painful it should be removed.

AFFECTIONS OF THE SEBACEOUS GLANDS

Adenoma Sebaceum Rhinophyma, or Lipoma Nasi.—Forms a bulbous hypertrophied mass on the skin of the nose

TREATMENT.—Excision followed by plastic repair.

Carcinoma Sebaceum causes a rodent ulcer (*see* p. 66).

TREATMENT.—(1) Excision of the whole thickness of the affected skin and subcutaneous tissue, with half-an-inch margin of healthy tissue; subsequent skin-grafting. (2) Repeated exposure to the X rays. This

Carcinoma Sebaceum—Treatment, ***continued.***

is suitable for inoperable cases, and those where removal would cause great deformity, or when operation is refused. (3) Radium. This acts much more rapidly and effectively than the X rays.

Sebaceous Cysts.—

OCCUR on any part of the body, but especially on the hairy parts, on the scalp behind the ears, or in the eyelids.

SIGNS.—A fluctuating swelling in the skin which moves freely over subjacent tissues. A blocked mouth of a sebaceous gland is often seen.

ANATOMY.—The cyst wall may be epithelial, or calcareous from degenerative changes (atheromatous), or it may present adenomatous growth. The contents are pultaceous, and consist of fat, epithelial débris, and and cholesterin. They may form a calcareous mass.

COMPLICATIONS —

INFECTION followed by suppuration.

SEBACEOUS HORN may grow up by superposition of successive layers of epithelium.

FUNGATING ADENOMA may follow its rupture.

DIAGNOSIS —

DERMOID CYSTS occur in special situations and always lie deep to the skin.

LIPOMATA are below the skin and are lobulated.

ABSCESSES have a more rapid course and affect the deeper tissues.

TREATMENT.—Dissect out

Mollusoum Contagiosum.—Yellowish-white umbilicated nodules about the size of a pea.

OCCURS on the face, or less often on other parts of the skin.

CONSISTS of a central core of epithelial cells supported by a fibrous stroma. Bodies like psorosperms are found, but are the result of cell degeneration.

They are locally contagious.

TREATMENT by excision.

CHAPTER XVII

INJURIES AND DISEASES OF NERVES

INJURIES OF NERVES

Causes.—

TRACTION DURING BIRTH
CONTUSIONS AND STRAINS
DISLOCATIONS AND FRACTURES } Producing rupture of some or all of the nerve-fibres.

PRESSURE: Growth of tumours—Aneurysm—Displaced bones (fracture, dislocation, cervical rib)—Inclusion in callus—Pressure of crutches or splints—Inflammatory products in bony canals

WOUNDS.

Effects of Total Division of a Mixed Nerve.—

ON PARTS SUPPLIED.—Paralysis of muscles Anæsthesia of skin: only partial, owing to the overlapping of sensory areas. Vasomotor paralysis, producing first hyperæmia then anæmia of parts. Glands do not respond to ordinary stimuli.

CHANGES IN THE NERVE—

Retraction of divided ends.

Formation of a hæmatoma, especially at the proximal end.

Organization of the hæmatoma into a bulbous nerve-end (traumatic neuroma). This contains bundles of new nerve fibrillæ embedded in fibrous tissue.

The peripheral end shrinks and atrophies

Wallerian degeneration occurs below the injury, and above as far as the first Ranvier's node. Begins at the fourth day after injury. Medullary substance breaks up into fat globules. Axis cylinders disappear in one month Nuclei of neurilemma proliferate Leucocytes invade and replace the nerve-fibres

CHANGES IN MUSCLES.—Muscle cells atrophy, or are replaced by fibrous tissue and fat. Deformities occur from unopposed action of the unparalysed muscles.

ELECTRICAL REACTIONS —Excitability by faradic current is rapidly lost. Excitability by galvanic current is at first increased, and lost only very slowly. The response to galvanism is a peculiar sluggish contraction called 'the reaction of degeneration'. A greater contraction occurs at the anode than at the kathode on closing the current. As long as any response to electrical stimuli remains there is hope of repair.

SENSORY CHANGES.—Peripheral sensory nerves are of three kinds physiologically.

1. NERVES OF DEEP SENSATION.—These perceive deep pressure and pain, and the movements and position of bones and joints. They probably run in the muscles, tendons, and bones, and their functions are seldom if ever lost by peripheral nerve division.

Effects of Total Division of a Mixed Nerve—Sensory Changes, ***continued.***

2. Protopathic Nerves.—These respond to painful skin impressions, e.g., a prick, and distinguish extreme temperatures. Their area of distribution is badly localized, and stimulation of them gives a widely radiating, tingling feeling. They are concerned with the production of reflex movements.
3. Epicritic Nerves.—These distinguish light touches, e.g., by a hair or brush, and also small differences of temperature. They convey a well-localized sensation.

In Division of a Sensory Nerve: (1) are unaffected, (2) are affected over a small and variable extent, as there is much overlapping of adjacent protopathic nerves, (3) are affected over a constant and well-defined area, considerably larger than that of (2). In a mixed nerve all three types of sensation are lost.

In Division of a Posterior Root there is always a larger loss of protopathic sensation than of epicritic. Thus the peripheral nerve is the unit of epicritic nerve-supply, and the posterior root is the unit of protopathic nerve-supply.

CHANGES IN THE JOINTS —A plastic synovitis occurs, resulting in ankylosis. Especially noticed in the small joints of the fingers.

TROPHIC CHANGES —

a. In Complete Division of the nerve without irritation or neuritis. Skin becomes rough, scaly, and œdematous Glands atrophy, and hence the skin is very dry Atrophy of the bones or cessation of growth occurs Hair and nails break, or fall off

b. When the Divided Nerve is Irritated (the commonest condition). Skin is thin, shiny, bluish red—'glossy skin' Vesicular and pustular eruptions occur. Chilblains are common Excessive sweat secretion. Ulcers and whitlows Hair falls out or breaks off. Nails are brittle and ridged, or lost by perionychia

c. In Both Cases.—Temperature falls as much as 8° F in affected parts

CHANGES IN THE CENTRAL NERVOUS SYSTEM —Spasms of a reflex nature, epileptic fits and dementia (both very rare)

RECOVERY OF A DIVIDED NERVE.—Can only occur when the two ends are brought close together, an inch separation being probably a maximum. The bulbous nerve-end offers great obstacles to repair.

New axis cylinders. (*a*) Grow down from proximal to distal part; (*b*) Develop in the distal segment.

Protopathic sensation returns first, and with it the tendency to trophic changes (blisters, etc) disappears. This takes place in periods varying from six weeks to one year

Epicritic sensation is the last function to be recovered, and the recovery is seldom, if ever, complete. It returns in six months to two years or more.

The time at which sensory recovery begins does not vary with the position of the lesion, but the time occupied by the recovery varies directly with the distance between the point of section and the periphery.

Motor recovery takes place first in the muscles nearest to the point of section, and eventually is more complete than the sensory recovery.

Tinel's sign of recovery after suture· Pressure over the nerve trunk causes tingling in the area of distribution of the nerve. As regeneration proceeds, the point where pressure produces this sign is further from the point of injury and nearer to the periphery.

Effects of Partial Division of a Mixed Nerve.—

MOTOR CHANGES.—Instead of the typical reaction of degeneration, there is, with failure of reaction to the faradic current, a ready and brisk reaction to galvanism, with no polar reversal.

SENSORY CHANGES.—Loss of epicritic sensation is the most marked and most constant sign.

A considerable portion of a mixed nerve can be divided, up to about one-third of all its fibres, without producing any appreciable effect.

Treatment.—

IF COMPLETE DIVISION is diagnosed.—

OPERATE immediately on all important nerves. Refresh the ends, and sew together. The divided ends of the nerve should be accurately trimmed and all ragged nerve-sheath removed. Suture the sheath only with fine catgut or thread, or fibrin sutures on an atraumatic needle. Nerve should be sutured without tension, and hæmostasis is important. Nerve handled gently and not allowed to become chilled during operation. Suture line protected by embedding in a flap of fascia or in amnioplastin. Recently fibrin plasma has been used to protect and reinforce the suture line If nerve-ends cannot be approximated, transposition of the nerve, or nerve anchoring and stretching followed by suture can be used. Prevent tension by tension sutures, and also by the position of the part.

SECONDARY SUTURE may be undertaken up to three years from the injury.

IF ONLY PRESSURE or partial laceration exists —

Wait to see whether natural recovery will take place.

Diagnosis of Nerve Division has to be made from :—

SPINAL CORD INJURY.—In this there is total loss of sensibility to either pain, heat, cold, tactile sensation, or deep sensation over a given area opposite to the side of the lesion, combined with a loss of movement and the sensation of passive movement on the same side as the lesion.

HYSTERIA.—In this there is a complete loss of all forms of sensation over an area the upper margin of which is a simple circle, constituting the so-called 'glove' or 'stocking' anæsthesia With this there may be a flaccid paralysis, but the electrical reactions are quite unchanged.

ISCHÆMIC PARALYSIS.—In this there may be areas of sensory loss with trophic changes, but the characteristic shortening of the flexor muscles without actual paralysis provides the clue

INFANTILE PARALYSIS

(*Anterior Poliomyelitis*)

Nature of the Disease.—A selective inflammation of the anterior-horn cells in the spinal cord, infective in character, due to the presence of the virus discovered by various workers but closely associated with the name of Flexner.

Course and Treatment.—The disease begins acutely with febrile symptoms, and in a few hours paralysis is evident and may affect any part or parts of the body. It affects most often the lower limbs, and may select one group of muscles and not another. It is a flaccid paralysis, with no sensory changes. In some cases the paralysis ascends and causes respiratory

Infantile Paralysis—Course and Treatment, *continued.*

paralysis. If the child is kept alive such paralysis will often recover, hence mechanical respirators such as Drinker's apparatus (*Fig.* 29) or the Bragg-Paul pulsator must be available. Three stages may be recognized:—

1. ACUTE STAGE.—Malaise. Fever. Paralysis. Hyperæsthesia. Pains in limbs.

   TREATMENT.—

   Serum.—Most authorities agree that when paralyses are present it is too late to give serum. This agrees with experiments on apes. There is experimental evidence that serum will negative or modify the disease in the pre-paralytic stage, but unfortunately diagnosis is seldom made before paralysis appears. In an epidemic serum may be given to children who develop a fever.

   Whole body to be fixed in bed or upon a padded frame. Limbs to be placed in light splints to maintain them in good position—i.e., with paralysed muscles relaxed. No massage or electrical treatment till all pain has disappeared from the limbs, which is usually about six weeks. movement during this stage produces a sympathetic congestion in the area of the cord affected, and merely increases and perpetuates the paralysis.

2. STAGE OF RECOVERY.—The paralysis, which is widespread at first, is largely recovered from, leaving isolated muscles or muscle groups paralysed.

   TREATMENT.—Massage and electrical treatment to affected limbs is usually given, but the value of electricity is disputed. Splints to prevent deformity Re-education in movements. Assisted active movements where gravity is abolished are invaluable.

3 STAGE OF PERMANENT PARALYSIS.—Certain muscle groups are finally paralysed, and unless prevented, their opponents will certainly

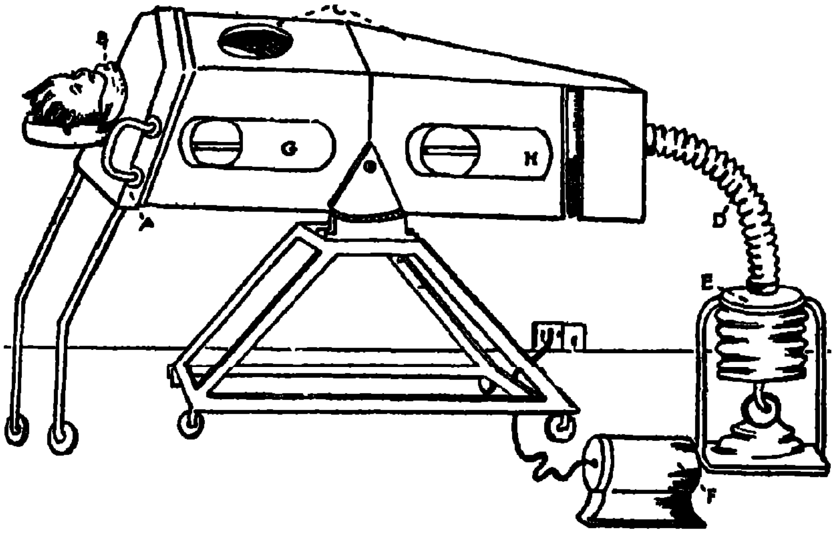

Fig. 29.—Drinker's apparatus. A, Upper part, which is attached to the bed and which can be withdrawn for putting the patient in or taking him out. B, Rubber collar, which makes the hole through which the patient's head emerges airtight. C, Lower part of apparatus into which the bed and headpiece slide. D, Pipe from air bellows. E, Air bellows. F, Motor working bellows. G and H, Trapdoors for access to the patient. (*From a photograph kindly lent by Sir Henry Gauvain.*)

distort the limbs. Deformities are due to the unbalanced pull of the healthy muscles. With proper treatment in the first and second stages there ought to be no contractures or deformities requiring correction.

TREATMENT may be of the following kinds:—

1. *Provision of splints*, e.g., jointed calliper for the leg, which will prevent deformity when the limb is used.
2. *Correction of deformities*—by tenotomy, or by weight traction, or successive plasters. The commonest operations are tenotomy of hip flexors, or knee flexors, or of the tendo Achillis.
3. *Arthrodesis or fixation of a joint* of which all the muscles are paralysed. The common example of this is fixation of the ankle or tarsal joints for flail foot.
4. *Transplantation of tendons.* This is only possible when there is a suitable healthy muscle to take the place of the paralysed one—e.g., the peroneus longus may be brought across the front of the foot to replace the tibialis anticus. (*See also* TALIPES, p. 170.)

NEURALGIA

Definition.—Pain in the area of a nerve distribution without any primary nerve lesion. Pain often recurs daily at the same time.

Varieties.—

1. SYMPTOMATIC OR SECONDARY —
   Toxic, e.g., malarial, gouty, lead.
   REFLEX, e.g., neuralgia secondary to carious tooth or glaucoma.
   PRESSURE, e.g., of aneurysm or new growth, on nerve-roots.
2. IDIOPATHIC OR PRIMARY.—Commonest in the fifth nerve.
   Also in Intercostals—Breast—Ovaries—Testes—Joints.

IDIOPATHIC NEURALGIA

With especial reference to Trigeminal Neuralgia

Aetiology.—Cause unknown. An ascending neuritis with arteriosclerosis has been suggested, but not demonstrated. Women more often than men. Generally after 40.

Symptoms.—

PAIN.—Paroxysmal attacks (epileptiform). The second or third division of the nerve is alone affected at first, but soon the attacks involve the other two divisions. First escapes longest. Each attack lasts only a few seconds to a minute. Pain during the attack is excruciating, sharp, and lancinating. Attacks at first are infrequent, but recur more often. Attacks are often brought on by sensory stimuli, e.g., cold draught, brushing the hair. Constant aching pain follows the paroxysm, and may last until the next.

SENSORY AND TROPHIC CHANGES.—Pressure over nerve trunks is very painful. Skin tender, and at places hyperæmic and œdematous. Profuse sweating, with lacrimation and increase of nasal discharge. Skin gets smooth and shiny; hairs fall out.

MOTOR SYMPTOMS.—Associated muscles thrown into reflex spasms or twitchings.

Treatment of Idiopathic Trigeminal Neuralgia.—

DRUGS.—Salicylates, quinine, iodides, croton chloride. All lose their effects rapidly. Avoid morphia at all costs.

Treatment of Idiopathic Trigeminal Neuralgia, ***continued.***

OPERATIVE.—

1. NERVE STRETCHING AND DISTAL NEURECTOMY give such temporary relief that they are not used now.
   Lingual neurectomy is often performed for cancer of tongue.
   Inferior dental nerve may be removed for toothache.
2. DIVISION OF THE SENSORY ROOT OF THE GASSERIAN GANGLION.—Permanent relief is afforded by section of the sensory root of the Gasserian ganglion.
   Frasier's Operation.—A vertical incision in the temporal region anterior to the pinna is used. Skull opened, dura of middle fossa raised; and Gasserian ganglion displayed. Sensory root divided, having been retracted with a small blunt hook. Complication is keratitis of eye of affected side, and for this reason a fractional division of the sensory root is better, dividing lower and outer two-thirds.
3. INJECTION OF THE BRANCHES OF THE NERVE with 80 per cent alcohol. This is done through a long needle thrust into the cheek so as to lie above the sigmoid curve of the jaw, and an endeavour is made to enter the foramen ovale or the sphenomaxillary fossa, in order to inject the actual nerve-trunks near the ganglion. Results are good for a year or more, and it can be repeated.
   This is the lateral approach. The anterior approach is also used as described in (4), only the nerve is injected and not the ganglion.
4. INJECTION OF GASSERIAN GANGLION.—The needle is thrust through the mouth in the angle between the upper jaw and the cheek, opposite the second molar tooth, until the base of the skull is struck. The point of the needle is guided into the foramen ovale, and about 1 c.c. of alcohol injected into the ganglion The effect will be more profound and more lasting than by the last method, but there is a danger of injecting the subarachnoid space and so damaging other nerves.
5. INTRAMEDULLARY TRACTOTOMY —Cerebellar approach and division of the pain fibres in the descending limb of the trigeminal tract in the medulla. Does not denervate the face and muscles of mastication are never paralysed.

AFFECTIONS OF SPECIAL NERVES

Optic Nerve.—

RUPTURES by fracture through anterior cranial fossa

COMPRESSED OR INFLAMED by intracranial diseases, especially meningitis and tumours.

INJURED by orbital growths, hæmorrhage, or cellulitis.

SYMPTOMS.—Blindness, optic atrophy, or optic neuritis.

Third Nerve.—

INJURED by fractures through sphenoidal fissure—Orbital tumours or aneurysms.

DISEASED by syphilitic disease of brain—i.e., meningitis.

SYMPTOMS.—Ptosis, or drooping of upper eyelid—External squint—Diplopia when looking inwards—Dilated pupil—Loss of accommodation—Slight exophthalmos due to the muscles holding the eyeball being flaccid.

Fourth Nerve.—Injuries and diseases as in the case of the third.

SYMPTOMS.—Squint and diplopia when looking downwards.

(For diseases of the fifth nerve, *see* TRIGEMINAL NEURALGIA, p. 143.)

Sixth Nerve.—Injuries and diseases as in the third.

SYMPTOMS.—Internal squint—Diplopia when looking outwards.

INJURIES AND DISEASES OF THE SPHENOIDAL FISSURE OR CAVERNOUS SINUS produce: Paralysis of all the muscles of the eye—Anæsthesia of the cornea and forehead—Venous congestion of the eye and conjunctiva.

OPHTHALMOPLEGIA EXTERNA, i.e., paralysis of all the external eye muscles, without congestion, is caused by syphilitic or tabetic disease of the floor of the third ventricle.

Seventh or Facial Nerve, or its centres, may be diseased or injured anywhere between the cerebral cortex and the branches of distribution.

INTRACRANIAL LESIONS.—

CEREBRAL CORTEX.—Injury—Pressure—Hæmorrhage. Part of the opposite side of the face paralysed.

CORONA RADIATA.—Hæmorrhage or thrombosis. Lower half of opposite side of face paralysed. Eyelids and occipitofrontalis (supplied by both sides of cortex) escape. Combined with paralysis of arm, and perhaps of leg.

IN PONS.—Hæmorrhage or growth. Paralysis, with atrophy of the same side of the face from affection of the facial nuclei. Paralysis of the opposite side of the body from affection of the pyramidal tract of the opposite side above the decussation of the pyramids.

NERVE-ROOT between the brain and the bone.—Injuries or tumours. Paralysis of the whole of the same side of the face. Generally associated with nerve deafness.

CRANIAL LESIONS.—

FRACTURE OF THE BASE OF THE SKULL running through the internal auditory meatus. Causing immediate laceration of the nerve, or later implication in callus.

OTITIS MEDIA causing compression of the nerve in the aqueductus Fallopii.

INJURY OF NERVE during mastoid operations.

A loss of taste in the anterior two-thirds of the side of the tongue follows a division of the nerve between the geniculate ganglion and the point where the chorda tympani leaves the trunk.

EXTRACRANIAL LESIONS.—Injury of the nerve by operations—Cold or inflammation—Tumours (especially malignant) of the parotid. Produce total facial paralysis.

FACIAL PARALYSIS.—

SYMPTOMS (total paralysis).—Eyelids cannot be closed. Eyeball is rolled up on attempting to shut eye. Corneal ulceration sometimes results from exposure. Epiphora from want of apposition of lower eyelid to eye. Face immobile and wrinkles smoothed out. Face drawn to opposite side. Cheek is flabby, and food collects between cheek and teeth from paralysis of buccinator.

PROGNOSIS.—In cerebral hæmorrhage—recovery generally takes place. In pontine hæmorrhage—atrophy occurs and recovery is rare (patient usually dies). In intra-osseous lesions—slow recovery is the rule. When the trunk of the nerve is divided paralysis may be permanent.

Seventh or Facial Nerve—Facial Paralysis, ***continued.***

TREATMENT.—Expectant in most cases. When paralysis is permanent: Grafting the hypoglossal or part of the spinal accessory nerve into the trunk of the facial.

In old cases benefit may be obtained by grafting a slip of fascia to form a sling to raise the drooping angle of the mouth.

Auditory Nerve and Its Centres.—Tumour or injury of the opposite temporosphenoidal lobe, fracture of the base of the skull through internal meatus, produce incurable deafness.

In certain cases of Ménière's disease section of the auditory nerve in its course in the posterior cerebral fossa is indicated.

Vagus.—

TRUNK is injured by: Fractures through the jugular foramen—Operations or tumours in the neck.

SYMPTOMS: Vomiting—Inhibition of heart's action—Palpitation—Laryngeal paralysis.

THE RECURRENT LARYNGEAL NERVE is injured by subclavian aneurysm on the right side—Aortic aneurysm on the left side—On both sides by injuries, operations, or malignant growths in the neck.

Produces laryngeal paralysis—especially abductor paralysis. Hoarse voice. Asphyxia if bilateral.

Eleventh or Spinal Accessory Nerve.—

INJURED OR DIVIDED BY:—

a. Fractures through the jugular foramen.

Produce paralysis of larynx and pharynx through affection of accessory portions. Paralysis of sternomastoid and trapezius (partial).

b. Operations in the neck, especially those for removal of glands.

Produce paralysis of sternomastoid (slight), and more complete of trapezius, with wasting and drooping of shoulder. If divided in posterior triangle only trapezius is affected.

SPASMODIC WRY NECK.—Clonic contraction of the sternomastoid and the small rotator muscles of the head. (*See* p. 163.)

Hypoglossal Nerve.—Injured by wound or operation in neck, or by carotid aneurysm or tumour. Produces unilateral paralysis and hemi-atrophy of tongue. The tongue when protruded deviates to the same side of the mouth.

Sympathetic Nerve in the Neck.—May be involved by tumours, or injured by stabs. Irritation produces dilatation of the pupil and widening of the palpebral fissure, with exophthalmos. Also unilateral sweating.

Paralysis produces contraction of the pupil, narrowing of the palpebral fissure, retraction of the eye, and dryness of skin.

Phrenic Nerve.—Arises from the third, fourth, and fifth cervical and goes to the diaphragm. Wounds in the neck may divide it, but unilateral division causes no symptoms. Irritation of the nerve is said to cause cough or hiccup.

Brachial Plexus.—

CAUSES OF INJURY.—

SUPRACLAVICULAR.—(*a*) Indirect violence. Excessive or sudden traction on the arm in attempting to save a fall, or at birth. (*b*) Direct injury. Cervical rib, fractured clavicle, or stab wounds. Usually affects the first dorsal.

INFRACLAVICULAR.—The various incidents during and after dislocation of the shoulder-joint.

DISTRIBUTION OF THE BRACHIAL ROOTS.—

THE POSTERIOR ROOTS.—Of these only two have well-defined areas of distribution, viz., the fifth cervical and the first dorsal, lesions of which cause a pre-axial and post-axial anæsthesia of the arm respectively.

THE ANTERIOR ROOTS.—

Fifth cervical—the deltoid, rotators, biceps, coraco-brachialis, brachialis anticus, supinators, and rhomboids.

Sixth cervical—pronators, radial extensors, clavicular part of pectoralis major, serratus magnus

Seventh cervical—triceps, ulnar extensors, finger extensors, lower part of pectoralis major.

Eighth cervical—flexors of wrist and fingers.

First dorsal—intrinsic muscles of the hand.

There are three common types of brachial plexus injury:—

1. WHOLE PLEXUS.—All the muscles of the arm are paralysed except the rhomboids and serratus. There are usually some signs of injury of the cervical sympathetic. In SUPRACLAVICULAR rupture sensation is lost over the whole arm except on the inner side adjacent to the axilla, which is supplied by the intercosto-humeral nerve. In INFRACLAVICULAR rupture the loss of sensation is complete.

2. UPPER ARM OR ERB-DUCHENNE TYPE (incomplete upper lesion).—Essentially a rupture of the fifth cervical anterior primary division. It is commonly caused by traction on the arm in a direction towards the feet. Paralysis of the deltoid, rotators, biceps, brachialis anticus, and supinators. The arm lies at the side, and the forearm is pronated. There is no loss of sensation Occasionally the deltoid and rotators alone are paralysed from a partial rupture of the fifth cervical in its upper part.

3. LOWER ARM OR KLUMPKE TYPE (incomplete lower lesion).—Is caused by a rupture of the eighth cervical and first dorsal nerve. Caused by traction on the arm upwards Paralysis of the intrinsic muscles of the hand, with sympathetic paralysis. Sensation is lost over the inner side of the arm and forearm.

THE INNER CORD OF THE PLEXUS may be injured by a subcoracoid dislocation. The symptoms are those of injury of the ulnar nerve, together with the small hand muscles supplied by the median (inner head of the median).

THE OUTER CORD OF THE PLEXUS is occasionally injured in dislocations. There will be paralysis of the biceps, coracobrachialis, and of all the muscles supplied by the median, except those in the hand.

THE POSTERIOR CORD is the most rarely injured part of the infra-clavicular plexus. The symptoms will be those of lesions of the musculospiral and circumflex nerves.

TREATMENT.—Open wounds are explored and the nerves sutured if possible. Subcutaneous injuries treated on expectant lines, and paralysed muscles put up in position of relaxation. Serious lesions in which the nerves are torn demand efforts at surgical repair, although the results are indifferent.

Cervical Rib.—

ANATOMY —The transverse process of the seventh—sometimes the sixth —cervical vertebra grows out and joins the rib below. May be partly

Cervical Rib—Anatomy, ***continued.***

rib and partly fibrous cord, or a fibrous cord only. In this course the cervical rib passes through the brachial plexus, and it may compress the lower part of this, together with the subclavian artery, between itself and the first true rib.

AETIOLOGY.—It is much commoner in females, usually bilateral; only a small proportion cause any symptoms, and these are then usually unilateral and on the right side. The symptoms first appear between the ages of twenty and thirty.

SYMPTOMS.—GENERAL WEAKNESS of the whole limb, noticed at the end of the day or after severe muscular exertion.

MUSCULAR WASTING, affecting chiefly the interossei, thenar, and hypothenar muscles.

PAIN shooting down the inner side of the arm and forearm into the ulnar side of the hand, also some tingling or spasticity of the inner fingers.

ALTERED PULSE. There is often a noticeable bruit over the subclavian artery, with a diminished pulse on the affected side. There may be cyanosis and coldness of the arm.

TREATMENT.—Until recently excision of the rib and its periosteum was the orthodox treatment. It has been found that division of the scalenus anticus muscle allows the rib to drop, and this much simpler operation gives very good results. Mild cases improve with physiotherapy to improve posture and muscle tone of the shoulder girdle.

Posterior Thoracic, or Nerve of Bell, which arises from the fifth, sixth, and seventh cervical nerves, and goes to the serratus magnus, is often paralysed by itself as the result of injury or neuritis.

SIGNS.—Winged scapula. Inability to raise the arm above a right angle.

The Circumflex (Axillary) Nerve may be injured by blows, by fractures of the surgical neck of the humerus, or by dislocations.

SIGNS.—Paralysis and wasting of the deltoid and teres minor, with anæsthesia over the lower two-thirds of the deltoid.

The Musculospiral (Radial) with its posterior interosseous branch is the most commonly injured of all the spinal nerves.

CAUSES.—Fractures and dislocations of the humerus and shoulder. Pressure of crutches at the posterior axillary fold. Pressure on the arm under the body during anæsthesia or a drunkard's sleep. Gunshot wounds. Fracture, usually gunshot, of the neck of the radius (affects posterior interosseous only).

SYMPTOMS.—Paralysis and wasting of the triceps, supinators, and extensors of the thumb, fingers, and wrist.

Wrist-drop is the most prominent sign, the hand being held in a position of pronation (*Fig.* 30). The fingers and thumb cannot be properly extended, but the terminal phalanges of the fingers can be extended by the interossei.

Anæsthesia.—If lesion is in upper third of arm (rare), there will be sensory loss over the back of the radial side of hand and thumb. If the lesion is in the lower third of the arm (common type), there is no sensory loss, owing to the fact that the external cutaneous branches of the musculospiral anastomose with the external cutaneous nerve, which in its turn anastomoses with the radial nerve. In the same way division of the radial nerve high up in the forearm produces no sensory loss.

If the lesion is below the elbow, the long supinator and the radial extensors will escape and there may be no wrist-drop, but only loss of extension in the thumb and fingers.

Treatment.—Whilst waiting for operative treatment, and during the whole period of recovery, a dorsiflexion ('cock-up') wrist splint (*Fig.* 31) must be worn, in order to prevent the extensor muscles becoming stretched.

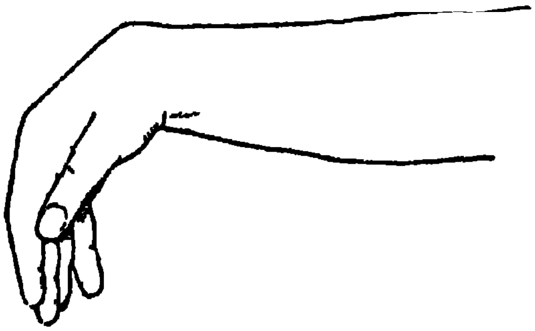

Fig. 30—Musculospiral nerve paralysis

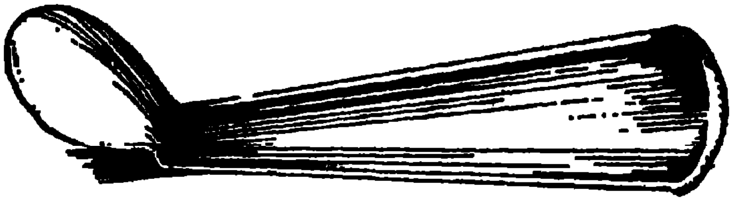

Fig. 31—The short 'cock-up' wrist splint

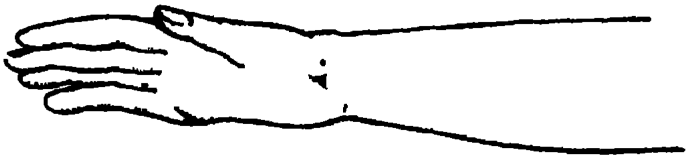

Fig. 32.—Median nerve paralysis—'ape hand'.

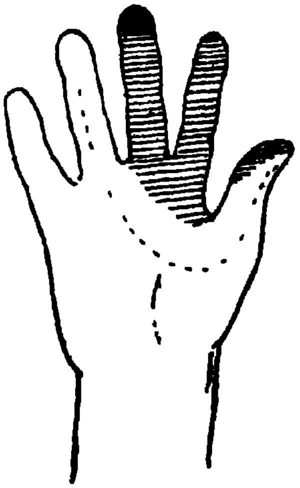

Fig 33.—Loss of sensation produced by division of median nerve. In this figure and in *Fig.* 36 the dotted line marks the limit of loss of sensation of touch, the shaded area that of pain

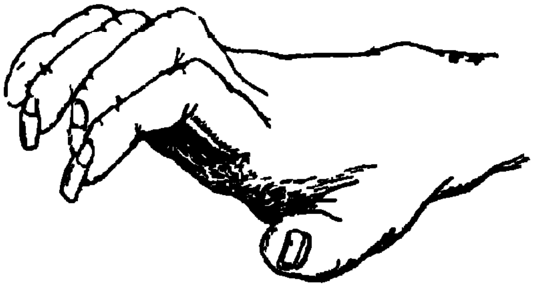

Fig. 34—Median and ulnar nerve paralysis.

Musculospiral Nerve Injuries—Treatment, ***continued.***

Tendon Transplantation.—When nerve suture has failed or is impossible, tendon transplantation gives a good result. From the flexor group the following muscles are isolated. flexor carpi radialis, pronator radii teres, palmaris longus, and flexor carpi ulnaris. These are cut as low down as possible and joined to: (1) The extensors of the wrist; (2) The extensors of the thumb; and (3) The extensor of the fingers.

Median Nerve.—Usually injured by wounds above the wrist.

SYMPTOMS —Paralysis and wasting of three short thumb muscles (the abductor, opponens, and part of the flexor brevis), with defective opposition of the thumb movement, also of the outer two lumbricals, which if the interossei are intact, give no sign (*Fig* 32).

The paralysis of the opponens pollicis is manifested by inability to bring the thumb across the hand parallel to the palmar surface It is difficult to detect, because the flexors and adductors together may simulate this movement

Anæsthesia (epicritic loss) of palmar aspect of thumb and adjacent two-and-a-half fingers, and of the dorsal aspect of the last, or last two, phalanges of the same (*Fig* 33) Trophic changes will be evident.

If the nerve is injured at the elbow or in the upper arm; Paralysis of both pronators, with loss of pronation, and of all the flexors, except part of the flexor profundus and the flexor carpi ulnaris

If the ulnar nerve also is divided, the deformity shown in *Fig* 34 results.

Ulnar Nerve.—

CAUSES OF INJURY.—Wounds or dislocations, especially in the neighbourhood of the elbow.

Delayed ulnar neuritis is seen in old fractures of the lateral condyle of the humerus, the nerve being stretched in the groove behind the medial epicondyle due to alteration of the carrying angle at the elbow

SYMPTOMS.—Paralysis of the lumbricals (inner two) and all the interossei, causing the *main-en-griffe*, i e, hyperextension of the metacarpo-phalangeal joints and flexion of the two inter-phalangeal joints (*Fig* 35). Wasting is seen between the metacarpals.

Paralysis of the adductor muscles of the thumb, and all the short muscles of the little finger, with wasting at the thenar and hypothenar eminences.

The paralysis of the adductors is shown by inability to bring the thumb against the index finger in a direction at right angles to the palmar surface. The long flexor or extensor may simulate this movement.

Paralysis of the flexor carpi ulnaris and part of the flexor profundus, with weakened hand-grasp and tendency to radial abduction of the hand

Anæsthesia (epicritic loss) of the little and half the ring fingers back and front. Also of the ulnar part of the hand (*Fig.* 36).

If divided just above the wrist, only the hand paralysis is seen, and there is no dorsal anæsthesia.

In all cases deep sensibility in the hand is lost only when the nerve is divided high up before it goes to the muscles, or when the flexor tendons as well as the terminal nerves are divided.

SPECIAL SPLINT FOR ULNAR AND MEDIAN LESIONS.—The clawed fingers should be gradually brought down by a combination of axial traction and flexion on a special Verrall's splint (*Figs.* 37, 38).

ANTERIOR TRANSPOSITION OF THE ULNAR NERVE may be required in delayed ulnar neuritis and in fractures of the internal epicondyle with irritation of the ulnar nerve.

The Cauda Equina may be injured by falls and blows apart from or together with injuries of the spine. The sensory loss is of the root type, i.e., the protopathic is larger than the epicritic loss, usually being limited to a saddle-shaped area on the buttocks. Spontaneous burning pain is common.

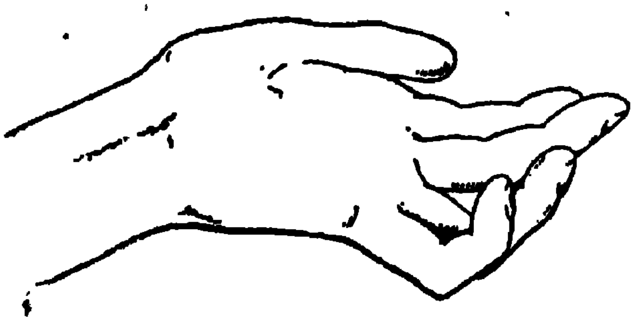

Fig. 35.—Ulnar nerve paralysis

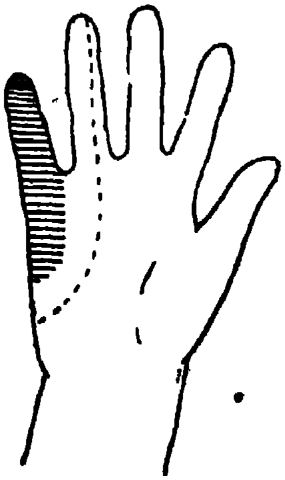

Fig. 36.—Loss of sensation produced by complete division of ulnar nerve.

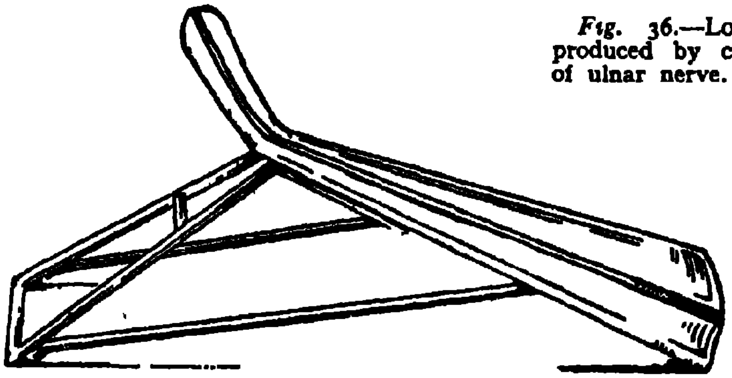

Fig. 37.—Verrall's splint.

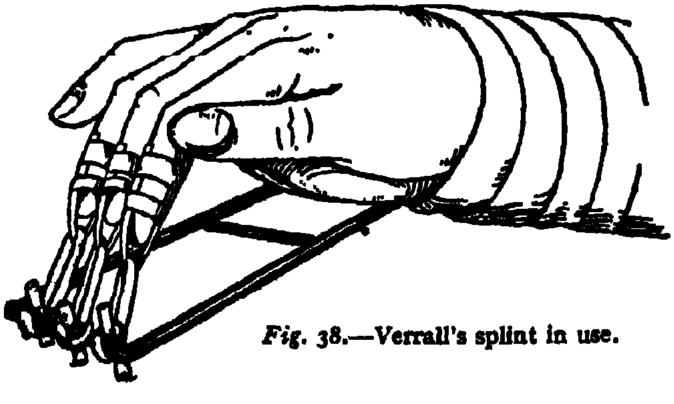

Fig. 38.—Verrall's splint in use.

Cauda Equina, *continued.*

DISTRIBUTION OF THE NERVES OF THE SACRAL PLEXUS.—

Fourth lumbar—the posterior muscles of the leg below the knee and the tibialis anticus.

Fifth lumbar—the anterior leg muscles (except the tibialis anticus) and the peronei.

Second sacral—the glutei and hamstrings.

Third and fourth sacral—the levator ani, sphincter ani, and perineum.

The Sciatic Nerve is very seldom injured, except by gunshot wounds, owing to its deep position and great strength. The signs are those of internal and external popliteal nerve injuries (*see below*), combined with paralysis of the hamstrings

The external division (external popliteal) is often injured, whilst the internal escapes In lesions of the whole nerve there is a very limited sensory loss on the inner side of the leg.

SCIATICA is neuralgia of the sciatic nerve.

CAUSES —(*a*) Doubtful nature, e g., cold, neuritis, rheumatism, gout, syphilis; (*b*) Pressure on the nerve, inside or outside the pelvis, by abscesses, aneurysm, or new growths; (*c*) Pressure on the nerve-roots by caries, new growth, or injuries of the spinal column or intervertebral discs; (*b*) Chronic spinal cord diseases, e.g , tabes.

SYMPTOMS —Pain down the back of the thigh, increased by pressure on the nerve, or by flexing the hip-joint when the knee is straight (stretches the nerve).

TREATMENT.—Drugs suitable for rheumatism, neuritis, etc. Absolute immobility for several weeks in plaster-of-Paris. Nerve stretching. Novocain and saline injections. Epidural injections. Oxygen inflation. Physiotherapy. (*See also* INTERVERTEBRAL DISCS, p. 289.)

External Popliteal Nerve.—May be torn or injured as it winds round the biceps tendon and neck of the fibula

SYMPTOMS.—*Anæsthesia* of the dorsum of the foot and *paralysis* of the extensor and peroneal muscle groups. *Talipes equinovarus* results.

TREATMENT.—Foot-drop must be prevented before nerve-suture and during recovery, by a toe-raising spring. When nerve-suture is impossible or has failed, the foot may be kept raised by making a hole in the crest of the tibia at its lower third and passing the tendons of the tibialis anticus and peroneus longus (cut from their muscles) through this hole, drawing them tight and sewing them in this position. Arthrodesis of the subastragaloid joint may be necessary to stabilize the foot.

Internal Popliteal Nerve.—Is seldom injured. Paralysis of the calf and flexor muscles of the toes, with some anæsthesia of the sole of the foot, and *talipes calcaneo-valgus.*

Anterior Crural Nerve.—Is seldom affected. Its injury causes paralysis of the quadriceps extensor muscle and sartorius, with flexion of the knee and anæsthesia of the greater part of the thigh and leg.

TREATMENT.—The flexor muscles of the knee may be brought forward to act as extensors, by attaching them to the patella. The biceps on the outer side, and the semitendinosus and gracilis on the inner, may thus be transplanted.

THE SYMPATHETIC NERVOUS SYSTEM

Anatomy.—Two sympathetic cords or trunks run, one on each side, from the base of the skull to the coccyx, lying on the side and front of the vertebral column. Each has ganglia upon it, three in the cervical portion, and one for each vertebral segment below (D 12, L 5, S 5). The ganglia are connected with the corresponding spinal nerves by white (medullated) and grey (non-medullated) rami communicantes. Nerves issue from the sympathetic ganglia to the plexuses of nerves situated along the course of the great blood-vessels and around the thoracic and abdominal viscera (cardiac, cœliac, solar, hypogastric plexuses). A sympathetic central nucleus exists in the region of the diencephalon.

PARASYMPATHETIC SYSTEM.—Originating in brain and sacral region, cranial parasympathetic fibres pass to the pupil, salivary glands, heart and alimentary canal, including also lungs. They are secretory and motor to the alimentary canal and inhibitory to the heart. The sacral portion of the parasympathetic is concerned with the "emptying processes."

Physiology.—The sympathetic nerves serve to innervate the unstriped muscle of the blood-vessels and viscera. They serve to maintain tone or tonic contraction of the vessels, of the voluntary muscles, and of parts of the viscera They are complementary or antagonistic to the action of the vagus nerves in the abdomen. Thus they cause spasm of sphincters at the pylorus and ileocæcal valve, but inhibit the action of the vagus in causing peristalsis of the bowel. They have an important role in the causation of pain, especially in the deep pain of viscera.

Indications for Operation on the Sympathetic Nervous System.—

I. VASCULAR DISORDERS.—Periarterial sympathectomy by removing the vasoconstrictor fibres allows dilatation of the vessels. Affects chiefly the smaller arteries. Indicated in: (1) Raynaud's Disease; (2) Buerger's Disease, (3) Acrocyanosis; (4) Erythromelalgia.

Before operation tests for vasodilatation must be performed by one of the following methods:—

1. *Local or Regional Anæsthesia* of affected sympathetic fibres, e.g., injection of stellate ganglion in the upper limb, and giving a spinal anæsthetic in the lower limb. The rise in skin temperature, etc., is ascertained, and the vasomotor index calculated.

   Vasomotor index =

   $$\frac{\text{Rise in skin temperature} - \text{rise in mouth temperature}}{\text{Rise in mouth temperature}}$$

   This should be at least 2·5 before sympathectomy can be of value.

2. *Hot-air Bath.*—Patient placed in a hot-air bath and the index calculated.

3. *Intravenous T.A.B. Technique* causing pyrexia.

Raynaud's Disease.—Affects mostly hands and feet, women more than men. Attacks precipitated by cold—fingers pale or cyanotic and cold, later red and tingling. Repeated attacks cause local organic changes. Treatment: Excision of stellate ganglion with removal of 2nd thoracic ganglion.

Sympathetic Nervous System—Indications for Operation, *continued.*

Buerger's Disease (thrombo-angiitis obliterans).—Mainly men, 30–50 years, lower limbs, high incidence among Jews. Intermittent claudication is an early symptom, also attacks of superficial phlebitis with thrombosis of the deeper vessels. Treated by lumbar ganglionectomy.

II. DISEASES OF ABDOMINAL VISCERA.—(1) Hirschsprung's Disease. (2) Renal Sympathetico-tonus. (3) Spastic Colon.

Hirschsprung's Disease.—Spinal anæsthesia has produced improvement in some cases, but not always with permanent results. Lumbar ganglionectomy produces the best results.

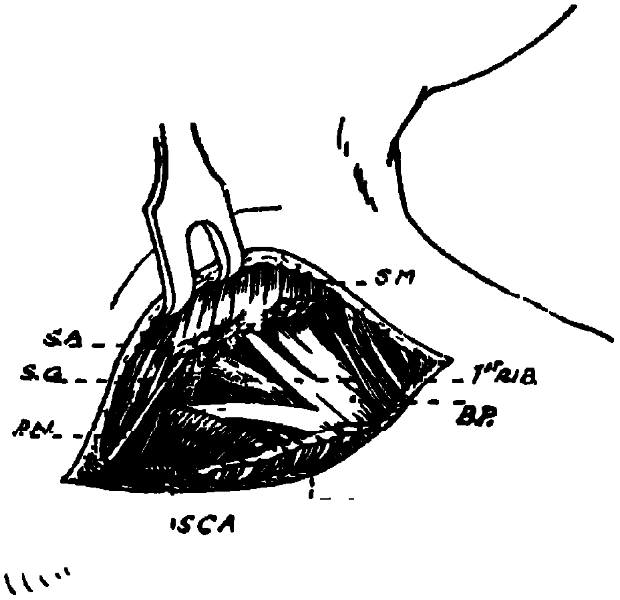

Fig. 39.—Exposure of stellate ganglion. S.M., Sternomastoid muscle (cut), B.P., Brachial plexus, S.C.A., Subclavian artery; P.N., Phrenic nerve, S.G., Stellate ganglion; S.A., Scalenus anterior muscle.

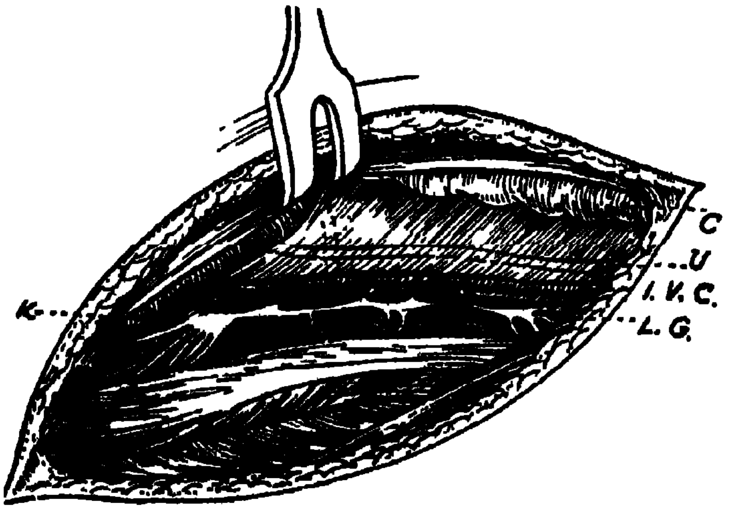

Fig. 40.—Exposure of lumbar ganglia. C., Colon; U., Ureter; I.V.C., Inferior vena cava; L.G., Lumbar ganglion; K., Kidney.

Renal Sympathetico-tonus.—There is a degree of hydronephrosis without any demonstrable cause, associated with renal pain, relieved by eserine. Treatment by renal denervation.

III. SYMPATHECTOMY FOR PAIN.—(1) Presacral Neurectomy in Dysmenorrhœa. (2) Presacral Neurectomy in Intractable Bladder Pain. (3) Trophic Ulcers. (4) Causalgia.

IV. SYMPTOMATIC GROUP.—

Scleroderma.

Anterior Poliomyelitis.—Sympathectomy improves the trophic skin changes.

Hyperidrosis.

Operations on the Sympathetic Nervous System.—

GANGLIONECTOMY.—This operation—i.e., the excision of one or more of the ganglia of the sympathetic chain—has the same effect as periarterial sympathectomy, but produces a much more lasting result. Ganglionectomy at the present time has fallen into two main groups: (1) Stellate ganglionectomy with excision of D1 and 2 for upper limb lesions (*Fig.* 39). (Usually performed through an anterior incision.) (2) Lumbar ganglionectomy with excision of L 2, 3, 4, 5, and rami for lower limb lesions (*Fig.* 40). (Usually performed through a renal incision or sometimes transperitoneal approach.) Rarely the superior cervical ganglion is removed, and recently attention has been devoted to the splanchnic nerves and ganglia in high blood-pressure.

RESECTION OF THE PRESACRAL NERVES.—For cases of intractable pain caused by cancer of the rectum or uterus and bad cases of dysmenorrhœa, the lower part of the aortic plexus and the nerves running down from it to the hypogastric plexus (presacral nerves) are resected.

PERIARTERIAL SYMPATHECTOMY is of value in certain cases that will not stand ganglionectomy, but only produces transient improvement. It will also relieve pain.

CHAPTER XVIII

AFFECTIONS OF MUSCLES, TENDONS, SYNOVIAL SHEATHS, AND BURSÆ

AFFECTIONS OF MUSCLES

Traumatic Affections of the Muscles.—

RUPTURE OF MUSCLE SHEATH.—Especially in the biceps cubiti and rectus femoris. Causes soft hernia-like protrusion of muscle fibre when latter contracts. Rarely necessary to operate.

DISLOCATION OF TENDONS.—Most common in the neck, long tendon of biceps, peroneus longus. Great pain and stiffness after some sudden exertion

TREATMENT is immobilization in plaster for 3 to 6 weeks in a position of complete muscular relaxation. Rarely (e g., with peroneus) an open operation to suture the ruptured ligaments and tendon sheath is desirable.

RUPTURE OR DIVISION OF MUSCLES AND TENDONS.—

1. CONTRACTION-RUPTURE.—Voluntary, from an excessive or ill-balanced purposive movement, e g , the rectus abdominis in labour. Involuntary, as in tetanus
2. TRAUMATIC —Contusions, e g., the extensor tendons of the fingers by a sharp blow over the knuckles. Wounds dividing muscles and tendons.

POSITION OF RUPTURE.—(*a*) In muscle belly; (*b*) At junction of belly and tendon; (*c*) In tendon; (*d*) At insertion of tendon, where a piece of bone may be broken off

MUSCLES COMMONLY AFFECTED.—Sternomastoid by traction during labour. Supraspinatus by slight violence, results in a painful shoulder and inability to raise the arm, may have nodule (calcification) in the tendon; treat by open operation, suture, abduction splint. Biceps cubiti (long head), frequently associated with osteo-arthritis of the shoulder-joint. Long extensor of the thumb may rupture in the after-treatment of a Colles's fracture. Flexor tendons of the fingers and thumb by stab and incised wounds Extensor tendons of the fingers by contusions Muscles of the back and rectus abdominis by violent strains, tetanus, and parturition. Tendon of the adductor longus in riding. The quadriceps tendon or the ligamentum patellæ by contraction. The inner head of the gastrocnemius, the tendo Achillis, or the plantaris in athletic exercises (tennis leg).

SIGNS.—Sudden sharp pain. Snapping sound. Loss of function. Swelling and ecchymosis, with a palpable gap if the muscle belly is affected.

RESULTS.—Recovery if treatment is good and the muscle belly affected. Permanent disability if the tendons are divided and retract. Stiffness from adhesions of the tendons to the sheath. Weakness from the stretching of a fibrous scar.

TREATMENT.—In slight cases, immobilization in a position of extreme relaxation of the affected muscle.

In most cases, union by suturing. If the gap is very long: (*a*) Turning back from each end a tendon flap; (*b*) Grafting the distal tendon into a neighbouring tendon with similar function; (*c*) Filling the gap by strand of catgut or a piece of tendon taken from elsewhere.

Inflammation and Degeneration of Muscles.—

1. SIMPLE MYOSITIS, resulting from a wound or contusion.
2. RHEUMATIC MYOSITIS, causing pain and stiffness, e.g., stiff neck and lumbago.
3. ACUTE SUPPURATIVE MYOSITIS arises by infection in pyæmia. Results in considerable cicatricial deformity.
4. CHRONIC TUBERCULOUS MYOSITIS, secondary to bone disease, e.g., psoas abscess
5. SYPHILITIC MYOSITIS from the formation of gummata, or fibroid thickening Most often seen in the sternomastoid.
6. PARASITIC MYOSITIS—TRICHINIASIS.—

The adult *Trichina* worm lives in the alimentary canal of pigs, rats, and other usually carnivorous animals It measures 1·5 mm. (male) to 3 mm. (female) long. Pigs become infected by eating dead rats, by being fed on offal, and from one another. The embryo worms work their way from the alimentary canal through the peritoneum to the muscles. The diaphragm, rib, and trunk muscles are most affected. When it reaches the muscle, the embryo becomes encysted between the fibres in an oval cyst 0·4 mm long. In this condition its vitality is very resistant to heat, cold, or pickling

CLINICALLY, the disease occurs in epidemics in Germany and America. Three stages are recognized: (*a*) A stage of enteritis with severe colic and diarrhœa, lasting about 10 days. (*b*) A febrile stage, temperature rising to 102–106° F., with severe muscular pains and swellings and some œdema; (*c*) A stage of subsidence, which occurs 6 weeks after infection. It is liable to be mistaken for enteric fever.

7. MYOSITIS OSSIFICANS.—

a. TRAUMATIC.—A muscle, after injury, in the process of repair becomes ossified. It then forms a hard, sharply-defined mass. This is seen most often in the adductor longus, and is known as 'rider's bone'. A more diffuse type of myositis ossificans is seen in the brachialis anticus above the elbow and in the deep parts of the quadriceps above the knee in injuries of these parts. The ossification creeps into the muscle from torn periosteum. The condition is caused or aggravated by ill-advised forced movements, especially in children, performed to prevent stiffness of an injured joint.

TREATMENT.—In the early stage the limb should be kept at rest. When mobility has to be restored, this should be done by slow and gradual extension or flexion without force.

In rare cases, and at a late stage when the disease is quiescent, improvement may be effected by dissecting out the plaques of bone from the deep muscle fibres.

Myositis Ossificans, *continued.*

b. IDIOPATHIC.—This affects many muscles, chiefly those of the back and trunk. It usually attacks young men, and is of slow and steady course, resulting in extensive ankylosis.

8. MYOSITIS FIBROSA, or the fibrous degeneration of muscles.

ISCHÆMIC PARALYSIS is the common name for this, which was first described as VOLKMANN'S CONTRACTURE.

CAUSES.—Pressure from within or without—e.g., effusion, displaced fragments, or tight splints—producing an interference with the vascular supply. There is usually both venous and arterial block. Recently it has been shown by Griffiths that there is a local spasm of the affected artery at the site of injury, and he advises exploration and resection of the affected artery.

POSITION.—It has generally been observed in the flexors of the forearm, and in the great majority of cases has followed fractures in the region of the elbow-joint.

PATHOLOGY.—There is a degeneration of the muscle fibres followed by a replacement fibrosis. Striation is lost.

Three Stages.—

First: At the time of vascular occlusion, and lasting for about forty-eight hours. Great pain, swelling, discoloration, blebs in the skin. Loss of pulse. Prompt relief of the causative pressure may bring relief.

Second: Whilst the muscles are undergoing degeneration. Lasts about six weeks. During this time proper splinting may prevent contractures, or peri-arterial sympathectomy may cause relief.

Third: Permanent degeneration of the flexor muscles with contracture.

SYMPTOMS.—

Flexion of the fingers at the phalangeal joints. These can only be extended when the wrist is flexed, showing that the flexor muscles are too short (*Fig.* 41).

Pronation of the hand is permanent from contraction of the pronator radii teres.

Some flexion of the elbow is caused by contraction of the flexor muscles which arise from the humerus.

Wasting is very marked, due to the atrophy of the muscles.

The bones of the forearm are ½ in. to 1 in. shorter than on the other side in cases where the condition has arisen in growing children.

Anæsthesia may be present, but has an irregular distribution; the hand is cold, the skin blue and shiny, but trophic ulceration is seldom seen.

Electrical reactions are generally merely diminished, but rarely the reaction of degeneration occurs. In this case, as in those showing anæsthesia and trophic lesions, the nerves have probably been affected as well as the muscles.

DIAGNOSIS must be made from:—

Primary Nerve Lesions, especially musculospiral paralysis. Here there is no flexor but an extensor lesion. In ulnar and median nerve lesions the paralysis is accompanied by anæsthesia, and both follow the anatomical distribution of the nerve. Reaction of degeneration is present.

Infantile Paralysis.—There is no local lesion. Reaction of degeneration is well marked, and contraction is much slower in its onset.

Synovitis and Injury.—In this, matting and contraction of the tendons may occur. The fingers cannot be extended when the wrist is flexed.

PROGNOSIS.—Is bad in proportion to the extent and duration without treatment of the affection. If the condition is recognized early, the deformity can be prevented or removed by systematic exercises.

TREATMENT.—

In the Early Stages.—

Some type of splinting which stretches the flexor tendons.

Resection of the portion of the artery in spasm.

In the Late Stages.—

Dividing the origin of the common flexor and pronator tendons from the medial condyle of the humerus.

Cutting and lengthening individual tendons.

Shortening both radius and ulna.

Results in late stages are not good.

Tumours of Muscles.—

PRIMARY.—Sarcoma, fibroma, rhabdomyoma. (Also gumma.)

Sarcoma is round- or spindle-celled. At first it forms a round encapsulated mass. But it grows rapidly and becomes diffused beyond the capsule. Wide excision or amputation is required. Any solid muscular swelling which grows steadily in spite of iodides should be regarded as sarcomatous.

SECONDARY.—Sarcoma and carcinoma.

Carcinoma of pectoralis major by secondary extension from carcinoma of breast.

DISEASES OF TENDON SHEATHS

Tenosynovitis.—

1. ACUTE SIMPLE.—From strains and sprains. Commonest in extensor muscles of the thumb and peronei of foot.
   SIGNS: Pain, swelling, and fine crepitus when the tendon is moved.
   TREATMENT as for a sprain.

2. ACUTE SEPTIC.—Caused by septic wounds or extension from neighbouring inflammatory foci. Condition is described on p. 29. Is apt to cause sloughing of the tendon and permanent adhesions unless treated early.
   TREATMENT.—By incisions and passive congestion.

3. SIMPLE CHRONIC.—Left after the acute attack, or caused by continuous over-strain. Increase of glairy effusion into the sheath, with swelling and weakness.
   TREATMENT.—By counter-irritation and firm pressure. Rarely by puncture or incision.

4. STENOSING TENOVAGINITIS.—This is probably a sequel of injury or chronic inflammation. It causes a painful snapping or catching of the digit when the affected tendon contracts. There is a painful swelling over the affected region. Common in the region of the wrist in the extensor muscles of the thumb.
   TREATMENT.—Incise the tendon sheath over the affected spot.

Tenosynovitis, *continued.*

5. CHRONIC TUBERCULOUS.—Common in the wrist and ankle. Two conditions may occur, separately or together:—
   - *a.* Great swelling and hyperplasia of the synovial membrane, in which tuberculous granulations develop.
   - *b.* Effusion of glairy fluid and deposit of fibrin in the form of 'melon-seed' bodies.

   SIGNS.—Chronic doughy swelling, with weakness and slight pain. May spread to the bones and joints, or suppurate.

   TREATMENT.—By rest and pressure. If this fails, open and scrape out, rubbing in iodoform.

Ganglion.—A chronic synovial cyst, generally connected with a tendon sheath, or a myxomatous degeneration of the tendon sheath.

OCCURS generally at the back of wrist, connected with thumb or 1st finger extensor tendons. May occur on front of wrist or in the ankle region.

CAUSED BY a hernial protrusion of synovial membrane through the tendon sheath.

CONSISTS OF a firm, round, elastic, painless swelling over a tendon, filled with fluid or colloid.

TREATMENT.—(1) Subcutaneous rupture by pressure; (2) Puncture followed by pressure; (3) Excision.

COMPOUND PALMAR GANGLION is a tuberculous synovitis which affects the synovial membranes of the flexor tendons of the fingers and thumb under the anterior annular ligament It forms a fluctuating swelling in the palm and above the wrist, which may extend up the thumb and little finger It contains: (1) Tuberculous granulations growing from the synovial membranes; (2) Some free fluid; (3) Fibrinous melon-seed bodies.

TREATMENT.—By rest, and firm pressure by immobilization in plaster-of-Paris; if this does not succeed, incision, scraping, and iodoform inunction. Permanent stiffness from tendon adhesions is likely to follow.

DISEASES OF BURSÆ

Bursitis.—

1. ACUTE SIMPLE BURSITIS.—From injury or irritation.

   TREATMENT —Rest, fomentations, and aspiration if necessary.

2. ACUTE SEPTIC BURSITIS.—From septic wounds, or infection of a simple bursitis.

   TREATMENT.—By incision and drainage.

3. CHRONIC SEROUS BURSITIS.—The bursa is enlarged and distended by chronic effusion. Very common in the prepatellar bursa, forming the 'housemaid's knee'; over the acromion, 'the deal-runner's shoulder'; over the ischial tuberosity, the 'weaver's' bottom'. It is caused by constant irritation and pressure. In the course of time the walls become thick and plastic or fibrous bursitis arises.

   TREATMENT.—(1) Pressure and counter-irritation—this is likely to succeed only temporarily and in thin-walled recent cases; (2) Pressure after aspiration—recurrence is the rule; (3) Aspiration and injection of iodine—uncertain and painful; (4) Excision—the only satisfactory treatment in really chronic cases.

4 CHRONIC PLASTIC BURSITIS —Inside, the sac becomes thickened by fibrous tissue and nodular masses, some of which may form free bodies

5 FIBROID BURSITIS —The sac becomes converted into a solid fibrous mass with small central cavity
TREATMENT for this and No 4 is only excision

6 TUBERCULOUS BURSITIS —Similar to synovial tuberculous changes elsewhere Usually leads to chronic suppuration
TREATMENT —Excision or scraping

7 SYPHILITIC BURSITIS —Very rarely in the form of a symmetrical serous affection in the secondary stage Common as a gummatous disease in the late stage, when it is apt to burst and cause the characteristic gyrate ulcers over the joint
TREATMENT —Treat by iodides, etc

8 GOUTY BURSITIS —Very common over the big toe, frequent over the olecranon Urate of soda forms a hard tophus which may lead to inflammation and discharge
TREATMENT —Constitutional treatment only

9 MALIGNANT BURSITIS —Rarely a bursa becomes the seat of a sarcomatous growth This is manifested by the nodular character and rapid growth of the tumour
TREATMENT —Free excision or amputation will be required

Special Bursæ liable to disease —

SUPRA-ACROMIAL —The 'deal-runner s shoulder'

SUBDELTOID, liable to be confused with shoulder-joint effusion (*see* Chap XXIII)

SUBSCAPULAR, may communicate with the joint and be affected in tuberculous disease of the articulation

SUPRA-OLECRANON —'Miner's elbow'

SUPRA-ISCHIAL —'Weaver's bottom'

GLUTEAL BURSÆ —(1) Between the osseous and fascial parts of the insertion of the gluteus maximus, (2) Between the great gluteal tendon and the great trochanter Causes abduction of the hip

SUB-PSOAS, generally bilateral, between the psoas tendon and the brim of the pelvis May communicate with the joint Causes flexion of hip, with pain on extension, other movements being free

PATELLAR BURSÆ (*Fig* 42) —These are four in number, though only one is commonly diseased —

1 Infrapatellar The bursa most commonly affected with all varieties of disease, constituting 'housemaid's knee'
2 Subquadriceps This usually communicates with the knee-joint and is affected with it
3 Supraligamentous, over the patellar ligament
4 Subligamentous, under the ligament, and may press into the joint itself

SEMIMEMBRANOSUS (*Fig* 42) —Between the inner head of origin of the gastrocnemius and the insertion of the semimembranosus It is tense on extension and lax on flexion It often communicates with the joint, and forms the commonest variety of 'Baker's cyst'

SUBSARTORIAL —Between the insertion of the sartorius and the inner tibial tuberosity, associated with the insertions of the semitendinosus and gracilis

Special Bursæ, *continued.*

Sub-achilles.—Forms swelling on either side of tendo Achillis.

Adventitious Bursæ.—Form over any normal or abnormal bony prominence. The commonest are over the vertebra prominens of the neck, between the hyoid bone and thyroid cartilage, the inner femoral condyle in genu valgum, the inner or outer sides of the foot in talipes valgus or varus, the metatarsophalangeal joint of the great toe in hallux valgus. The last is known as a Bunion.

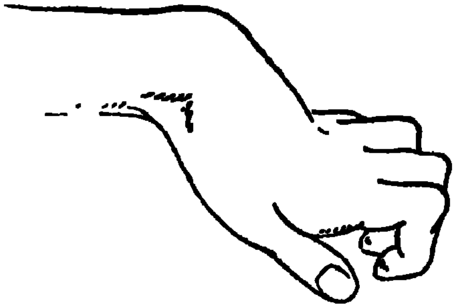

Fig. 41.—Ischæmic paralysis.

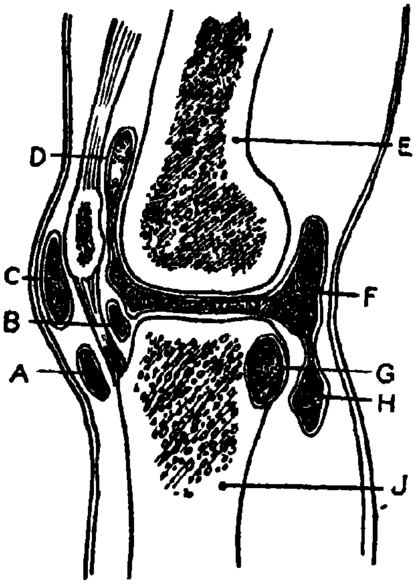

Fig. 42.—Diagram of the knee in longitudinal section, showing the position of various synovial cysts and bursæ. A, Bursa in front of ligamentum patellæ; B, Bursa behind ligamentum patellæ, C, Prepatellar bursa; D, Synovial pouch beneath quadriceps tendon, E, Femur; F, Posterior extension of synovial cavity, G, Bursa beneath semimembranosus tendon; H, Downward and backward extension of synovial cavity—i.e., a Baker's cyst; J, Tibia.

CHAPTER XIX

DEFORMITIES

Torticollis—Wry Neck (*Fig.* 43.)—

ANATOMY.—Contraction of sternomastoid (constant); of trapezius, deep posterior cervical muscles, cervical fascia (often). Head inclines to affected side. Face turns to unaffected side. Atrophy of affected side of the face. Primary curve of cervical spine. Secondary curve of dorsal spine. Secondary shortening of ligaments and moulding of bones and joints.

VARIETIES.—

CONGENITAL.—Generally following difficult labour, producing kinking of vessels and nerves of sternomastoid. Hæmatoma of sternomastoid is formed with a late myositis fibrosa. Microscopically identical with Volkman's contracture.

ACQUIRED.—

Traumatic —From partial dislocation of cervical spine.

Rheumatic —From myositis.

Spastic.—Clonic spasms of affected muscles; due to reflex or central irritation of cortical centres; young adults, generally women, with epileptic history

Reflex.—Generally due to cervical caries.

Paralytic.—From paralysis (infantile generally) of the opposite muscles.

Neurotic.—In hysterical women

DIAGNOSIS from CARIES OF SPINE (tenderness and pain on movement). DEEP ABSCESS of the neck. RHEUMATIC INFLAMMATION. By absence of pain, and tense band formed by the sternomastoid.

TREATMENT —

SLIGHT CASES.—Massage, retentive apparatus.

CASES WITH WELL-MARKED CONTRACTURE —Tenotomy of sternomastoid and fascia Open incision across origin of sternomastoid Free division of the muscle and any bands of fascia. Retention apparatus for a few weeks. Massage and exercises

SPASTIC CASES.—First try general treatment.

Operation: Excision of parts of spinal accessory of affected side, and posterior primary divisions of upper three cervical nerves on the opposite side. Results are poor, as cortical changes may be present.

Cervical Rib.—*See* p. 147.

Scoliosis.—Lateral curvature of the spine, with rotation of vertebræ.

CAUSES.—

CONGENITAL.—From presence of a half vertebra.

RICKETS.—Early or late.

INFANTILE PARALYSIS.—Affecting the back muscles.

Scoliosis—Causes, *continued.*

STATIC CAUSES (to compensate for another deformity).—Torticollis—Empyema—Hip disease or dislocation—Any shortening of the lower limb.

ADOLESCENCE.—Anæmia and asthenia during rapid growth. Muscles and ligaments stretch. Especially in occupations with much standing, lifting, or carrying.

ANATOMY.—

VERTICAL CURVES.—Usually main curve with convexity to the right occupying the dorsal region. Secondary curve to the left in lumbar region.

ROTATION.—Due to greater displacement of bodies than of the laminæ of the vertebræ.

Bodies will be directed to the convexity, spines to the concavity of the curve.

The ribs will project on the right side posteriorly, and be flattened on the opposite

The ribs and the breast will project on the left side anteriorly.

The sternum remains in the mid-line, but is twisted to the right.

Right shoulder projects behind and is raised ('shoulder grows out').

Left hip appears more prominent.

SECONDARY CHANGES.—Vertebræ and discs become shorter, and the ligaments and muscles are shortened, on the side of the concavity. Joints become ankylosed.

STAGES OF THE DEFORMITY.—

1. The deformity can be rectified by the patient's own muscular effort.
2. Can be rectified when the spine is flexed, or by manipulation, e.g., suspension.
3. Deformity becomes fixed by the shortening of muscles and ligaments and by ankylosis.

DIAGNOSIS.—Primary deformities or diseases of shoulder, breast, or hip must be distinguished from similar deformities secondary to scoliosis.

PROGNOSIS depends on the stage the deformity has reached.

When it can be reduced by manipulation the prognosis is good.

When it cannot be altered it cannot be cured, but may be arrested.

TREATMENT.—Remove the cause in static cases. Treat general conditions, e.g., rickets or anæmia.

IN ADOLESCENT CASES.—

Rest on an inclined plane.

Massage and cold douches.

Voluntary 'redressement': correcting deformity in front of a mirror.

Exercises: Flexion and extension of the back when standing. Horizontal bar exercises. Flexion and extension of the back when lying face down at the end of a table.

Carefully applied spinal support. This is not an absolutely rigid jacket, but a steel support which allows some spinal movement, and by the pressure of pads and springs tends to correct the deformity.

ABBOTT'S TREATMENT.—The patient is slung in a kind of canvas hammock which allows the spine to lie in a flexed position. Bands are passed round the trunk in such a manner that traction on these reduces the deformity, both lateral and rotatory. In this partly corrected position a plaster jacket is applied. Windows are cut in the jacket, and through these pads are inserted from time to time, so as further to press upon the convexity of the curves. After a few months the jacket is renewed and a further degree of correction obtained.

Kyphosis.—Abnormal backward curve of the spine. Usually limited to the dorsal region, unless it follows organic spinal disease.

CAUSES.—There are three groups of cases:—

1. DEFECTIVE GROWTH OR HABIT.—Rickets in children—Adolescence—Occupations which necessitate stooping—Senile atrophy of muscles of the back.
2. GENERAL DISEASE OF THE SPINE AND ITS MUSCLES, ETC.—Osteo-arthritis—Osteitis deformans—Osteomalacia—Acromegaly—Pulmonary osteo-arthropathy.
3. LOCAL SPINAL DISEASE.—Fractures—Pott's disease—New growths.

TREATMENT (in the first group only).—

Remove the cause, if possible (e g., myopia and any stooping habit).

Massage, electricity, and exercises for the back.

Recumbency on a hard, flat mattress or board.

Chance's splint: this consists of two steel bars fixed to a pelvic girdle and lying by the side of the vertebral column. To these are fixed adjustable padded plates which exert constant pressure on the protuberant parts of the spine and ribs, without preventing mobility.

Lordosis.—An abnormal forward curve of the spine, usually in the lumbar region.

It is always secondary to: (1) Diseases of the hip or of the psoas muscle. (2) A primary kyphotic curve above. (3) Any abnormal growth which places the centre of gravity forwards, e g., pregnancy.

Spondylolisthesis.—Forward and downward dislocation of the fifth lumbar vertebra on the body of the sacrum (*Fig.* 44). It is commoner in men than women. Always associated with defective development of the arches and

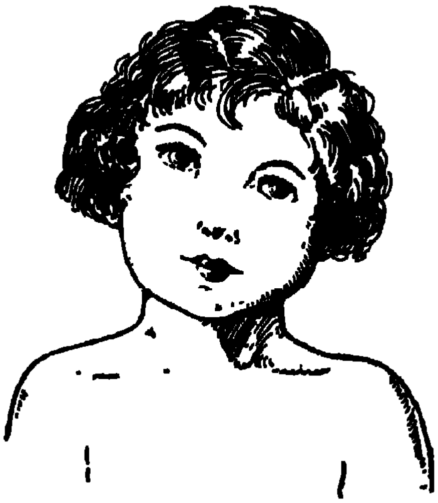

Fig. 43.—Torticollis. Left sterno-mastoid is contracted. Head bent to the left, chin turned to the right. Note the asymmetry of the face.

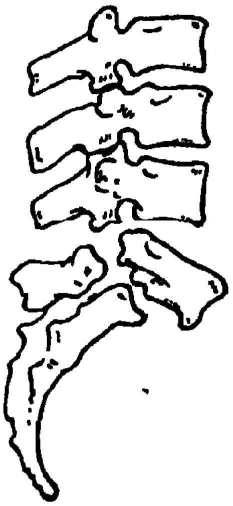

Fig. 44.—Spondylolisthesis.

Spondylolisthesis, *continued.*

articular processes of the fifth lumbar vertebra, which allows the latter to slip over the top of the sacrum. Causes characteristic deformity of deep depression of the lower lumbar spine and protuberant buttocks. Severe pain of the 'low backache' type.

TREATMENT.—Preliminary traction on both legs to try and reduce deformity. Followed by bone-grafting. *Posterior operation* is difficult and inefficient because the posterior part of the fifth lumbar vertebra is defective. *Anterior operation* consists in driving an autogenous graft from the tibia from the fifth lumbar into the first sacral vertebra. Watson Jones suggests double hip spica applied while the patient is suspended with both hips in flexion.

Deformities of the Upper Extremity.—

SPRENGEL'S SHOULDER.—Congenital deformity. The vertebral border of the scapula is raised; there is often a bar of bone or cartilage connecting the scapula to the spine.

TREATMENT.—By removal of the latter.

CLUB-HAND —Usually associated with congenital absence of the radius.

MADELUNG'S DEFORMITY OF THE WRIST.—Lower end of ulna projects upon dorsum of wrist. Styloid of radius is higher than that of ulna The hand is adducted, and in most cases displaced forwards (rarely backwards). It occurs at the ages of 8–18, and is much commoner in girls than boys. It is probably due to a partial arrest of development in the radial epiphysis Treatment consists in a cuneiform osteotomy of the radius performed when growth has ceased, associated with shortening the ulna.

SYNOSTOSIS OF RADIUS AND ULNA.—Usually the upper part of radius is deficient and fused with the ulna.

TREATMENT.—Cut away the upper part of radius, and cover stump with fascia. Prognosis unsatisfactory

POLYDACTYLISM.—Supernumerary fingers or toes: usually only affects the phalanges.

ECTRODACTYLISM —Absence of one or more digits.

MACRODACTYLISM.—Overgrowth of one or more digits.

SYNDACTYLISM.—Fusion of adjacent digits, either complete or only at their bases—the so-called webbed fingers or toes. The same condition may be acquired after burns.

TREATMENT.—By a plastic operation.

CONGENITAL CONTRACTION OF THE FINGERS.—Usually of the little finger only. The last two phalanges are flexed, the first hyperextended. The central digital prolongation of palmar fascia is contracted.

TREATMENT.—By the division of this band.

SPRING- OR SNAP-FINGER.—Development of a sesamoid bone in the flexor tendon, which catches under the tendon sheath opposite the metacarpo-phalangeal joint.

MALLET- OR HAMMER-FINGER.—Flexion of the terminal phalanx. Acquired deformity from rupture of the extensor aponeurosis.

TREATMENT —By a palmar splint or suture of the tendon.

DUPUYTREN'S CONTRACTION.—An acquired flexion of the fingers.

Usually in GOUTY men; often SYMMETRICAL, often HEREDITARY.

CONSTANT PRESSURE on the palm by an instrument may cause it.

Affects the ring or little finger first, the former more often than the latter; the others later.

The flexion is of the first and second phalanges, the third is extended.

An indurated nodule forms in the palmar fascia.

Consists of a contraction of the palmar fascia and its lateral prolongations into the digits. Associated with thickening and contraction of the skin and contraction of the ligaments of the interphalangeal joints.

DIAGNOSIS from congenital contraction (above) and contraction of the flexor tendons, which are relaxed on flexing the wrist.

TREATMENT.—

1. Multiple subcutaneous division of the fascial bands, followed by patient splinting, though in early cases splinting may arrest the deformity.
2. Careful excision of the affected palmar fascia. Probably a skin defect will be left, and this must be treated by grafting.

N.B.—In all old-standing cases, complete cure is almost impossible, whilst relapse after partial cure is the rule.

Congenital Dislocation of Hip.—*See* Chap. XXII.

Coxa Vara.—Deformity of the hip-joint due to a lessening of the angle between the neck and shaft of the femur (*Fig.* 45).

The angle is normally about 125°; at birth it is much more open (160°), and continues to diminish until growth is complete at 18–20. In late life atrophy of the neck may lead to further closing of the angle.

CAUSES AND VARIETIES.—

RICKETS.—In young children. Bilateral. Very rare.

ADOLESCENCE —The common form. The normal closure of the angle of the neck of the femur is carried too far.

Carrying heavy weights, and late rickets may also be concerned in this form.

EPIPHYSIAL COXA VARA.—Males 14–16 years, partial or complete downward displacement of epiphysis of head of femur. Cause is obscure. Found in heavy, over-weight, and under-sexed child, and thought to be part of a pituitary dyscrasia Rickets and staphylococcal infection have been suggested as possible causes. Trauma may be a factor, but the contralateral epiphysis has been known to become displaced in a patient under treatment for the opposite side.

SENILE FORM.—Atrophy of the neck makes the bone yield.

Usually associated with osteo-arthritis.

ANATOMY.—The angle between the neck and shaft is reduced to a right or even an acute angle. The neck of the bone is shorter than normal. The margins of the head are overgrown, The back of the femoral neck is more absorbed than the front, so that the shaft is everted.

SYMPTOMS.—Aching and tiredness, with marked limp. Shortening of the leg, the trochanter rising above its normal position and becoming very prominent. Eversion of the leg at the hip-joint. Adduction very marked, especially on flexing the thigh. Scissor-leg deformity if both sides are affected. Abduction and internal rotation very limited. Other movements of the hip are free and painless. There is a marked absence of tenderness on pressure.

Coxa Vara, ***continued.***

DIAGNOSIS.—From: Tuberculous disease of hip (q.v.)—Congenital dislocation (q.v.)—Mal-united fracture.

TREATMENT.—

Rest in Early Stages when deformity is still in progress, with traction in the line of the deformity until this is reduced, and later a walking calliper is worn for 6 months.

Subtrochanteric Osteotomy, after growth has ceased. The limb should be put up in an abducted position. Later a tilting of the pelvis will make up for much of the shortening.

Coxa Valga.—A rare condition. The opposite to vara, i.e., the femoral angle is more widely opened, and varies from 130° upwards to 180°.

CAUSES.—

Congenital —Frequently found in congenital hip disease. It may be the cause or result of the dislocation

Acquired.—Any hanging limb (e.g., paralysis) or stump will acquire a straight-necked femur. Infantile paralysis, scoliosis, or other static causes. Mal-union after fractures.

May be unilateral or bilateral. In the latter case it is probably a slight degree of congenital dislocation.

SYMPTOMS AND SIGNS.—

Limping towards the affected side: the gait is rolling in bilateral cases. Lengthening of the limb by 2–3 cm

Abduction and external rotation of the leg, whilst adduction is limited. Region of the trochanter is flat, and the trochanter is below Nélaton's line. Radiograph shows the straight neck.

TREATMENT.—By raising the shoe on the opposite side. Subtrochanteric osteotomy if the deformity is serious.

Genu Valgum—Knock-knee.—

CAUSES.—

Rickets, the common cause in young children.

Static Causes, i.e., long standing in a rapidly-growing patient whose ligaments are soft and relaxed.

Traumatic Conditions which interfere with the position or growth of the condyles or tibial tuberosities.

ANATOMY.—One or more of three bony changes have occurred:—

1. The lower end of the femur is bent outwards.
2. The upper end of the tibia is bent inwards.

These two conditions are most common in rickets.

3. The internal condyle of the femur and internal tuberosity of the tibia are developed disproportionately to the external parts. The common condition in adolescents.

The internal lateral ligament is relaxed, and in many cases the internal tibial tuberosity can be separated from the femur by forcible abduction.

The external lateral ligament, iliotibial band, and biceps tendon are proportionately shortened.

The patella is displaced towards the outer side of the knee.

PATHOLOGY.—In standing, especially with the feet apart, the line of the tibia lies outside that of the femur, the internal lateral ligament of the knee is relaxed, the external half of the joint is pressed together, the internal half is pulled apart. Hence the internal ligament stretches and the internal parts of the joint grow faster than the external.

SIGNS.—

When the knees are extended and touching each other the feet are widely separated.

With very slight flexion of the knees the ankles come together in the majority of cases. This is due to the fact that flexion is always accompanied by some internal rotation which swings the feet round towards one another.

The toes point outwards when standing. This is due to an exaggerated external rotation.

When walking the knees are kept slightly flexed, so as to minimize the deformity, which, as above explained, tends to disappear on flexion.

A bursa often forms over the inner sides of the knees when they knock together.

It is usually bilateral. Rarely it may be unilateral, or one knee may be in genu valgum and the other genu varum.

COMPLICATIONS —Scoliosis or flat-foot often coexists with genu valgum. The pelvis is tilted in unilateral cases.

TREATMENT.—

1. Rest with Splints.—In all rickety cases—In all patients below the age of puberty—In unhealthy patients when the condition is progressing.
   - Young children should be kept off the ground, either in bed or by application of splints extending beyond the feet.
   - Long outside splints should be re-applied daily, with massage night and morning.
   - Starch bandages should be used only to fix the splints for one or two weeks at a time at first, to get the child used to them, or in cases where no one can be trusted to re-apply them.
   - Iron splints fixed into the boots, with straps to fasten above and below the knee, for adolescent cases.
2. Special Boots.—In all ambulant cases, or those in pre-operative stages, the boots should be well raised on the inner sides so as to invert the feet and take the strain off the internal lateral ligaments of the knees.
3. Operation.—For all cases in which growth has ceased and in which the deformity is permanent.
   - a. *Osteotomy of the Shaft of the Femur* (Macewen's operation).— The bone is partly cut and partly broken just above the condyles. The limb is put up in a rectified position. This is the easiest and best operation in most cases
   - b. *Cuneiform Osteotomy of the Tibia.*—A wedge-shaped piece is cut out from the shaft of the tibia on its inner side below the tubercle. This is probably the most accurate method, but it requires some precision.

Genu Varum—Bow Legs.—Is similar, but opposite to the above. All the changes on the inner side of the joint above described are here external, and *vice versa*. It is much rarer than genu valgum. It seldom requires treatment, as it causes less inconvenience.

TREATMENT when required is on the same lines as the above, femoral osteotomy being the operation of choice.

TALIPES (CLUB FOOT)

Causes.—

CONGENITAL.—Often hereditary. Malposition in utero. Reversion to a simian type.

ACQUIRED.—

NERVE DISEASE.—Anterior poliomyelitis. Cerebral and spinal sclerosis producing spastic paralysis. Lesions of peripheral nerves.

MUSCULAR DISEASE.—Suppuration and contraction in muscles and fasciæ.

BONE INJURIES.—Fracture and dislocations with bad union about the ankle.

COMPENSATORY to other deformity.

STATIC CAUSES.—Prolonged standing—Decubitus.

Varieties.—

TALIPES EQUINUS.—Hyperextension of ankle. Walks on the toes (*Fig.* 46).

TALIPES CALCANEUS.—Flexion of the ankle. Walks on the heel(*Fig.* 48).

TALIPES VARUS.—Adduction of the foot at midtarsal joint. Inner margin of the sole raised Walks on outer side of foot.

TALIPES VALGUS.—Abduction of the foot at midtarsal joint. Outer margin of the sole raised Walks on inner side of the foot (*Fig.* 49).

TALIPES EQUINO-VARUS (*Fig.* 47), CALCANEO-VALGUS.—The commonest combinations of the above

TALIPES EQUINO-VALGUS, CALCANEO-VARUS.—Rare.

General Features of Congenital Talipes.—Exists from birth. May be associated with other anomalies as hare-lip, spina bifida. Often bilateral. Is associated with primary malformation of bones and ligaments and contraction of muscles. Except in early infancy the deformity cannot be corrected by simple manipulation No wasting or spasm of muscles. No trophic changes or defects in the circulation. Electrical reactions of the muscles are unaltered Growth of the limb is not impaired. Furrows are formed in the flexures of the foot.

TREATMENT.—

IN INFANCY.—Daily manipulation, massage, and bandaging.

AFTER THE CHILD HAS BEGUN TO WALK.—The foot is wrenched into overcorrected position and put up in plaster for some weeks. A metal brace is worn for a time until the child can be taught to walk correctly.

IN CHILDHOOD.—When deformity cannot be improved by manipulation: Division of all tendons and ligaments on the inner aspect of the midtarsal joint. Of these the tibialis posticus and the calcaneo-scaphoid ligament are the most important. Forcible correction of the deformity, followed by plaster. Fixing in corrected position by plaster. Massage and passive movements. Osteotomy.

IN ADULT CASES.—Osteotomy. Removal of a wedge of bone from the outer margin of the foot.

General Features of Paralytic Talipes.—Begins in second or third year with infantile paralysis. Seldom bilateral. Limb is cold, blue, and clammy. Muscles are much wasted and paralysed. Electrical reaction absent in paralysed muscles. General growth of the bones diminished. The limb is flabby, and can easily be restored to the correct position until late contractions have developed. Furrows do not form in the flexures.

TREATMENT.—

In Early Stages.—Serum is of doubtful value. Lumbar puncture if meningeal signs are present. Limbs put in light splints to maintain the correct position. Complete rest until all pain has gone—usually about six weeks; then massage and electrical treatment. Re-education in movements. Splints to correct attitude—i.e., paralysed muscles must be relaxed.

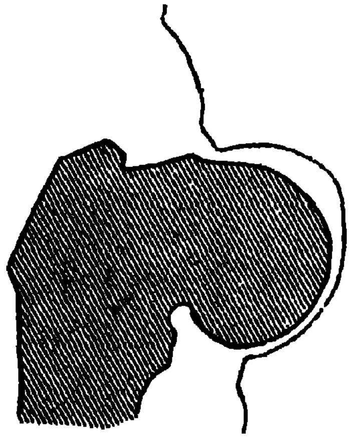

Fig. 45.—Coxa vara. The angle between the neck and shaft of the femur is reduced to a right angle.

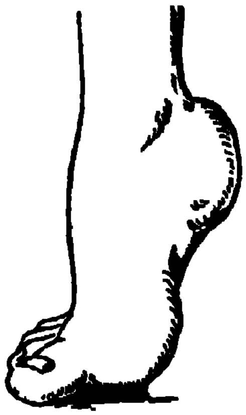

Fig 46.—Talipes equinus.

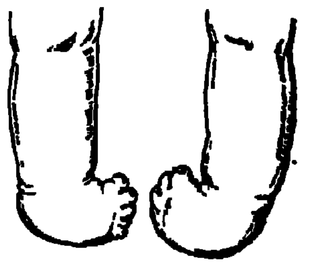

Fig. 47.—Congenital talipes equino-varus.

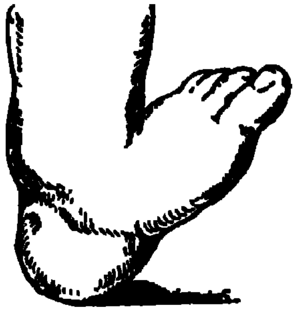

Fig. 48.—Talipes calcaneus.

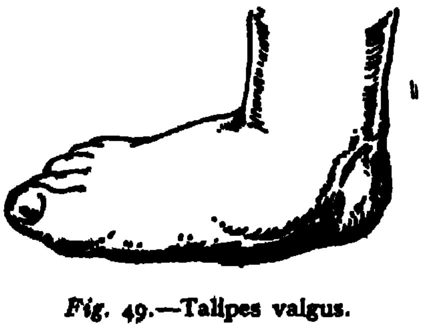

Fig. 49.—Talipes valgus.

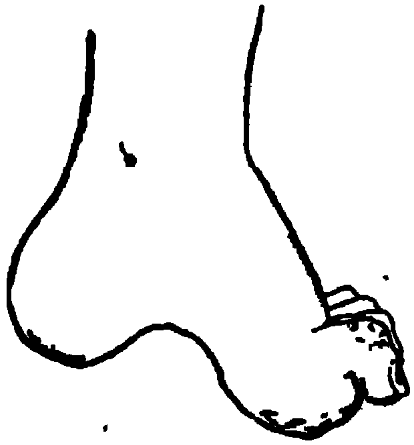

Fig. 50.—Pes cavus.

Paralytic Talipes—Treatment, ***continued.***

IN CASES WHERE NO FURTHER MUSCULAR RECOVERY IS POSSIBLE.—

Where Deformity has not been Prevented :—

Tenotomy of muscles opposed to the paralysed groups.

Grafting parts of unparalysed tendons into those which are paralysed.

For Flail Foot.—If anterior muscles are paralysed, and there is foot-drop, a bone block is fitted to the back of the ankle with the foot raised. If the posterior muscles are paralysed, and heel depressed, an anterior bone block is used. If the whole of the front of the foot is flail, then the midtarsal bones and joints are excised and fixed.

A special boot with irons and springs is useful in cases where operation has not been successful.

General Features of Spastic Talipes.—The symptoms of spinal sclerosis or cerebral disease are present The deformity can be reduced by manipulation, except in very old cases. Exaggerated knee-jerks and ankle clonus. Muscles do not atrophy till late.

TREATMENT —Re-education of muscles. Some retentive apparatus. Massage and electrical treatment strictly contra-indicated. Stoffel's partial neurectomy of the affected nerve trunks is of value.

Talipes Equinus.—

CAUSES —Nearly always acquired. Infantile paralysis of anterior tibial muscles Partial paralysis of all leg muscles, the calf muscles overcoming the anterior. Spastic contraction of the calf muscles. Compensatory to any shortening of the leg From pressure of the bedclothes.

SIGNS —Three degrees of extension of the ankle:—

1 Foot cannot be flexed beyond a right angle. Thus the toes catch the ground in walking.

2. The heel cannot be brought to the ground. Patient walks on the balls of the toes (*Fig.* 46).

3. Foot is in a line with leg or a little behind it. Toes are doubled under the foot, and patient walks on their dorsal surfaces.

Pes cavus is often present from a flexion of the sole at the mid-tarsal joint, especially in paralytic cases.

TREATMENT.—Tenotomy of the tendo Achillis. Plaster-of-Paris in over-corrected position.

IN PARALYTIC CASES.—Grafting half tendo Achillis into the tibialis anticus, and the peroneus longus into extensor tendon. A boot with toe-raising spring, or a bone block behind the ankle.

COMPENSATORY TALIPES EQUINUS should not be treated unless the primary condition is cured.

Talipes Equino-varus.—

CAUSES.—

CONGENITAL.—Represents a reversion to a simian type. In infants the head and neck of the astragalus form an angle of 35° inwards with the body. If this persists or is exaggerated, adduction of the foot with varus results.

ACQUIRED.—Paralysis of extensor and peroneal muscles. Tibialis anticus may escape or not. Over-action of calf muscles and tibialis posticus, and sometimes of tibialis anticus. Injury of the external popliteal nerve (*see* p. 152).

SIGNS.—Heel cannot be brought down to the ground. Foot is adducted at the mid-tarsal joint. Patient walks on the outer margin of the sole (*Fig.* 47). A transverse furrow crosses the sole at the mid-tarsal joint, and a longitudinal furrow runs along the sole in congenital cases. A callosity or bursa is formed over the cuboid. A secondary contraction occurs of plantar fascia.

ANATOMY.—

ASTRAGALUS is misshaped. The head is set at an angle of 50° with the body. It is displaced forwards and inwards from the ankle-joint.

SCAPHOID is on the inner side of the astragalus and almost touches the internal malleolus.

THE LIGAMENTS on the sole and inner side of ankle are contracted, viz.: Internal lateral ligament, inferior calcaneo-scaphoid ligament, plantar fascia.

THE FOLLOWING MUSCLES ARE CONTRACTED: Tibialis posticus, tibialis anticus, flexor longus hallucis, flexor longus digitorum, abductor hallucis, tendo Achillis.

TREATMENT.—

IN SLIGHT OR EARLY CASES—Massage, manipulation, and bandages. Malleable splints.

IN CASES WHERE CONTRACTION OF THE MUSCLES AND LIGAMENTS HAS OCCURRED.—

1. Tenotomy of tibialis posticus and anticus Division of internal lateral ligament in its anterior part. Division of the plantar fascia and spring ligament. These divisions of tendons and ligaments may be done in stages, getting as much correction and fixation in plaster after each as possible. Or they may be done all at once by an open operation.

   Subsequent tenotomy of tendo Achillis.
2. Removal of the whole or anterior part of the astragalus, with shortening of the peroneal tendons and displacing the foot backwards. A special boot is required to accommodate the long heel. Although this sounds formidable, it is probably the best treatment in all bad cases in patients over two years.

IN CASES OF OLD STANDING, WHERE ANKYLOSIS AND BONY CHANGE ARE MARKED, or where other measures have failed.—Excision of astragalus, or wedge-shaped tarsectomy from the outer side of foot. Dunn's triple arthrodesis in old neglected cases.

IN PARALYTIC CASES.—After correcting the deformity by tenotomies, graft half the tendo Achillis into the peroneus longus, and the tibialis anticus into the peroneus brevis.

Talipes Calcaneus.—May be either congenital or paralytic. Sometimes follows a too free tenotomy of the tendo Achillis. The patient walks on his heel (*Fig.* 48), the gait is heavy and stamping. The extensor tendons are contracted. The tendo Achillis is stretched or the posterior muscles are paralysed.

Talipes Calcaneus, *continued.*

TREATMENT.—

In Congenital Cases.—Tenotomy of the extensor tendons.

In Paralytic Cases.—Grafting peroneus longus tendon into tendo Achillis. Boot with a toe-depressing spring.

In Traumatic Cases.—Shortening of the tendo Achillis, or transplanting the tuberosity of the os calcis lower down.

Talipes Valgus.—Rare except in conjunction with flat-foot. May be congenital or paralytic. There is abduction of the foot at the mid-tarsal joint and an eversion of the sole of the foot (*Fig.* 49). The peronei muscles are contracted in congenital cases. The tibial and some of the extensors are paralysed in paralytic cases.

TREATMENT.—Division of the peronei tendons in congenital cases. Grafting of part of the tendo Achillis into the tibialis posticus in paralytic.

Flat-foot—Pes Planus.—In a simple form consists in a mere loss or flattening of the arches of the foot, but it is nearly always combined with some degree of valgus, i.e , of abduction and eversion of the front part of the foot.

CAUSES.—

Congenital —Diminution in the normal angle of neck and body of astragalus predisposes to flat-foot.

Acquired.—

1. *Static.*
2. *Traumatic* —Fractures of foot Sequel of fracture-dislocations at the ankle-joint.
3. *Inflammatory.*—Rheumatic, gonococcal.
4. *Paralytic.*—Paralysis of tibialis posticus and intrinsic muscles of foot.
5. *Spastic.*—Spasm of the peroneal muscles.

VARIETIES.—

Actively Mobile.—This is a normal foot, the arch of which can be fully formed by the patient's own muscular action, as in standing on tip-toe. It is seen in many healthy children, ballet dancers, and negroes. Requires no treatment.

Passively Mobile.—Arch can be restored by manipulation but not by the patient's own effort. Treat by muscle training and wedged shoes.

Immobile.—The arch is lost, and there is pain on attempting to correct. Usually results from foot-strain. Treat by forcible manipulation under an anæsthetic.

ANATOMY.—

1. The Longitudinal Arch of the foot is flattened. The muscles, ligaments, and tendons in the sole are stretched, viz.: the inferior calcaneoscaphoid or spring ligament, the calcaneocuboid ligaments (plantar ligaments), the tendons of the tibialis posticus and anticus, the flexores longi digitorum and hallucis, with the short muscles.

   The giving way of the calcaneoscaphoid ligament allows the astragalus to drop, so that it may actually touch the ground.
2. The Transverse Arch is flattened, the foot being broader than normal.
3. Abduction of the Foot, chiefly at mid-tarsal joint. The astragalo-scaphoid joint projects prominently on the inner side of the sole.

4. Eversion of the Sole of the Foot.—This also takes place at the mid-tarsal joint. The inner margin of the sole is depressed, the outer raised. The peronei tendons are tense and rigid.
5. Eventually, in old, neglected, and inflammatory cases, the bones become distorted by osteophytic outgrowths, and the joints, especially the astragaloscaphoid, obliterated by ankylosis.

SYMPTOMS.—Walk is shuffling or waddling. The toes are turned outwards (splay-feet). The heels do not leave the ground before the toes. The whole sole comes into contact with the floor. All power of jumping or dancing or raising the body on the toes is lost or diminished. Aching pain on the sole of the foot over the mid-tarsal joint is present, much worse on standing or walking.

DEGREES OF MUSCULAR AND ARTICULAR CHANGE.—

1. A Mere Loss of Strength and Tone in the Muscles, the ligaments being stretched. The patient can rectify the deformity to a great extent when he places his toes together and stands on tip-toe. The deformity can be easily rectified by passive movements.
2. Actual Paralysis or Functional Incapacity of the muscles, with contraction of the peronei The patient cannot stand on his toes, or in any way restore the arch of his foot Passive movements will, however, rectify this deformity.
3. Adhesions and Contractions fix the deformity so that neither active nor passive movements have any effect on it.
4. Actual Bony Deformity and Ankylosis render the condition permanent.

TREATMENT —

1. For the First Degree.—Massage, with tip-toe exercises. Boots with stiffening on the inner side of the uppers and thickened inner border to the sole.
2. For the Second Degree.—In addition to the above, Whitman's metal spring and a boot with an outside leg iron with a valgus T-strap.
3. For the Third Degree, especially in inflammatory cases.—Tenotomy of the peronei (where necessary). Wrenching the foot into good position under an anæsthetic. Fixing in a corrected position in plaster-of-Paris. A boot with a leg-iron must be worn for some time.
4. For the Fourth Degree.—Some form of tarsectomy. Removal of a wedge-shaped piece from the inner border of the sole. Removal of parts of the astragalus or scaphoid, or both.

Pes Cavus, or Claw-foot.—The longitudinal arch of the foot is exaggerated (*Fig.* 50). There is some degree of hammer-toes. It usually accompanies a slight degree of talipes equinus. There is more or less pain and disability on walking. May occur in nervous diseases as Friedreich's ataxia; also with spina bifida. Also follows poliomyelitis and trauma. Is essentially a deformity of the forepart of the foot—this dislocates downwards at the mid-tarsal joint. Followed by hyperextension of the toes at the metatarso-phalangeal joints and hammer-toes result.

TREATMENT.—Tenotomy of the tendo Achillis (in equinus cases). Fasciotomy of the plantar fascia. Boot should have a low heel and be provided with a transverse bar below and behind the heads of the metatarsal bones.

Pes Cavus, or Claw-foot—Treatment, *continued.*

TRANSPLANTATION OF TENDON of the extensor proprius hallucis. This tendon is cut near to its insertion, and is passed through a hole in the neck of the 1st metatarsal bone, thus raising the dropped metatarsal. The tendons of the extensor longus digitorum may be similarly treated in the other toes. Also Steidler's muscle slide operation.

In severe grades where all the toes are hammer and there is some equino-varus. In these cases, in addition to fasciotomy and tenotomy of the tendo Achillis, the astragalus should be removed, or a triple arthrodesis performed, and, in the most extreme cases, the toes also with the heads of the metatarsal bones should be removed.

Hallux Valgus.—Abduction of the phalanges with adduction of the metatarsal of the great toe. It is often associated with flat-foot, the transverse arch being flattened and the first metatarsal turned inwards. Is often caused by pointed-toed boots. The metatarso-phalangeal joint becomes very prominent, and is usually affected with hypertrophic osteo-arthritis. A marked bunion, i e , a chronically inflamed bursa, forms over the prominent joint. The toes are crowded together, hammer-toe often coexisting in the other toes (*Fig.* 51).

TREATMENT.—A straight inner-edged boot with a toe-post from the sole to keep the toe in position, for early cases

A spring along the inner side of the foot drawing the great toe inwards by a band.

Excision of the joint and the bunion is the best treatment of most cases. A flap of fascia is prepared from the inner aspect of the toe and is fixed over the neck of the metatarsal bone after excision of its head.

The external metatarsal condyle may be separated, and then slid back and pegged, after cutting the internal lateral ligament.

Hallux Rigidus or Flexus.—A flexion of the first phalanx on the metatarsal of the great toe The joint is affected by osteo-arthritis. It often accompanies flat-foot.

TREATMENT is first that of the flat-foot, and then if necessary an excision of the base of the proximal phalanx (Keller operation). A metatarsal bar is helpful in the early stages.

Hammer-toe.—Hyperextension of the first phalanx, with flexion of the other phalanges. The first interphalangeal joint forms an upward projection. The point of the toe is on the ground. Corns or bursæ are formed

Fig. 51.—Hallux valgus.

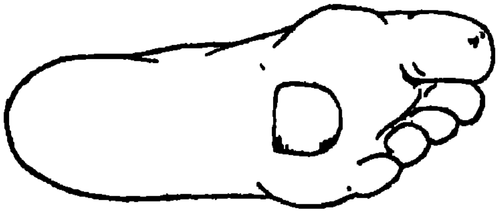

Fig. 52.—Pad for splayed foot.

under the head of the metatarsal bone and the point of the toe, and over the interphalangeal joint. The second toe is most frequently affected. The extensor tendons stand out very prominently.

CAUSES.—Sometimes congenital. Secondary to talipes equinus and pes cavus, or hallux valgus. Paralysis of the interossei and lumbricales. Short boots with high heels and pointed toes.

ANATOMY.—Partial dislocation of the second phalanx downwards. Marked shortening of the lateral digital bands of plantar fascia and of the lateral ligaments of this joint.

TREATMENT.—Excision of the head of the first phalanx. Hibbs' 'spike' operation.

A plantar splint to which the toe is bound down may be used in slight cases or after operation.

Metatarsalgia, or Morton's Disease.—Pain at the heads of the metatarsal bones. Probably a variety of flat-foot affecting chiefly the transverse arch. Possibly a direct pressure on the digital nerves results It occurs specially in gouty or rheumatic subjects. The foot is broader than normal, and corns form on the sole beneath the heads of the metatarsal bones.

TREATMENT.—Rest and massage. Support by some kind of a valgus pad (*Fig.* 52) or brace.

In severe cases, a ligature round the necks of the metatarsal bones, bracing them together. This ligature is best formed by a strip of fascia lata from the patient's own leg.

CHAPTER XX

FRACTURES

Predisposing Causes.—

AGE, SEX, and OCCUPATION.—

Young children, in whom ossification is incomplete, and who are constantly falling.

Old people, in whom some atrophy of the bone takes place.

Labouring classes, athletes, etc , because of the activity of their occupation.

Male sex. Except lower end of radius and neck of femur.

MORBID BONE CONDITIONS.—

GENERAL.—Atrophy: senile, disuse, paralysis—Fragilitas ossium—Rickets—Osteomalacia.

NERVE DISEASES.—Tabes, syringomyelia, general paralysis.

LOCAL BONE DISEASES.—Sarcoma—Carcinoma—Myeloma—Gumma—Caries or necrosis: septic, tuberculous, syphilitic—Fibrocystic disease.

Exciting Causes.—

DIRECT VIOLENCE.—Transverse in direction. Little displacement. Often compound or comminuted.

INDIRECT VIOLENCE —Oblique or spiral in direction. Great displacement.

MUSCULAR ACTION.—Patella and olecranon, common. Other bones, rare.

Spontaneous Fractures.—Cases where the exciting cause is trivial and fracture determined by a pre-existing morbid bone condition. These are:

1. FRAGILITAS OSSIUM.—The commonest cause in children. An imperfection in the ossification, the only symptom of which is the liability to fractures. It manifests itself in children, who lose the tendency in adult life. It may occur in new-born children, where the fractures are usually multiple. Union is rapid and normal.
2. NERVE CONDITIONS.—Tabes, syringomyelia, general paralysis. Tabes is the commonest cause in adults. There is no demonstrable change in the bone; possibly the ataxia may be concerned in it. The fracture is remarkably painless. It unites rapidly if properly immobilized, but there is an exuberant callus formation.
3. ATROPHY.—The common cause in old people. May be senile or due to disuse or paralysis. The hard bone is replaced by fat-containing cancellous tissue.
4. OSTEOMALACIA.—Usually attacks women, and is then associated with pregnancy. The tendency to fractures is associated with a softening and bending of the bones.
5. SCURVY AND RICKETS.—Usually occurs at the epiphysial junction in scurvy, and in the shafts of the long bones in rickets.

6. INFLAMMATORY BONE DISEASES, whether they produce caries or necrosis. Tubercle (commonly), septic and syphilitic disease (both rarely).
7. TUMOURS OF BONE.—
   - *a.* Sarcoma, usually primary, may be medullary or periosteal. The fracture will not unite.
   - *b.* Carcinoma—always secondary. The fracture usually unites if it is immobilized.
   - *c.* Myeloma—often associated with Bence-Jones' protein in the urine.
   - *d.* Gumma.
   - *e.* Fibrocystic disease.
   - *f.* Cysts, simple or hydatid.

Varieties of Fracture.—

CLOSED (simple).—No communication with a surface wound.

OPEN (compound).—Fracture communicates with a surface wound.

INCOMPLETE FRACTURE.—Bone not broken through.

Greenstick.—Occurs in children, especially with rickets. Concavity of long bone is bent. Convexity is splintered.

Fissures of long bones, especially near joints.

Crushing of outer table of bones: Occurs in skull—Small cancellous bones.

COMPLETE FRACTURES may be—

Transverse, especially when caused by direct violence.

Oblique, especially when caused by indirect violence.

Spiral, especially when caused by rotation.

Comminuted, when bone is broken into more than two pieces.

Impacted, when one fragment is driven into another.

Complicated, when combined with an injury of another important structure, e.g., artery or joint

SEPARATION OF EPIPHYSES.—Common in patients under twenty. May occur up to twenty-five.

Causes.—Joint injuries which produce dislocation in older patients. Syphilis in infants. Tubercle. Infective osteomyelitis

Anatomy.—Fracture runs through diaphysial side of the ossifying cartilage. Fractured surface of the epiphysis is concave. The older the patient, the more likely is it to involve the shaft as well as the epiphysial line. Periosteum closely attached to epiphysis is stripped off shaft. Shaft may be thrust through the periosteal sleeve.

Results.—(1) Recovery as in fracture, if apposition is good; (2) Retarded length of a long bone—deformity of wrist or ankle resulting from this; (3) Suppuration, with death of epiphysis; or (4) Arthritis from extension to joint.

Epiphyses Affected, in order of frequency:—

Upper and lower ends of humerus, lower end of radius, lower end of femur, about equal.

Upper end of femur; upper end of tibia, tibial tubercle alone, great trochanter, rare.

Signs of Fracture.—

1. WOUND, CONTUSION, OR BRUISING.
2. UNNATURAL MOBILITY in the shaft of long bone.

Signs of Fracture, *continued.*

3. CREPITUS on movement.
   - May be Simulated by: Osteo-arthritis—Tenosynovitis—Surgical emphysema.
   - May be Absent in: Fracture with impaction—Separated epiphysis—Wide separation of fragments—Interposition of soft parts—Partial fracture (greenstick).
4. DEFORMITY or displacement.
   - Caused By: (*a*) Violence; (*b*) Weight of limb; (*c*) Muscular action; (*d*) Manipulation.
   - Varieties.—Angular—Longitudinal: overlapping or separation—Lateral—Rotatory—Depression.
   - Evidences of Displacement.—Altered line of the bone—Altered measurement (generally shortened)—Altered relation of neighbouring bony points.
5. PAIN and loss of function.
6. RADIOGRAPH showing fissure or fracture.

Symptoms following Fractures.—

Shock, slight in uncomplicated cases.

Febrile reaction. Aseptic traumatic fever. Temperature 100° to 101° F. on second and third days. In proportion to ecchymosis.

Rare Symptoms.—

Delirium tremens in drinkers.

Fat embolism: Dyspnœa—Syncope—Coma (from blocking of capillaries of lungs and brain with fat globules).

Complications of a Fracture.—May cause:—

1. INFECTION: Non-union, necrosis, or deformity.
2. JOINT INVOLVEMENT: Arthritis and adhesions.
3. DISLOCATION.
4. INJURY TO MAIN ARTERY: Aneurysm or gangrene.
5. INJURIES OF VEINS: Ecchymosis and œdema.
6. INJURIES OF NERVES: Rupture or involvement in callus.
7. INJURY TO VISCUS.

Repair of Fractures.—

FIRST WEEK —

Blood-clot forms round the broken ends and beneath the torn periosteum. Soft parts become swollen and œdematous.

SECOND WEEK.—

Granulation Tissue replaces the clot. This is complete about the tenth day, when the fracture is fixed in a mass of granulation.

Calcification of the granulation tissue to form callus. Medullary tissue is also absorbed and replaced by callus. The ends of the bone become porous.

Callus is situated:—

1. Between periosteum and bone = external callus.
2. Between bony fragments = permanent or intermediate callus.
3. As a medullary plug = internal callus (*Fig.* 53, A).

Calcification occurs first in the external, then in the internal, and last in the intermediate callus.

Amount of callus is increased by movement and irritation.

THIRD WEEK TO EIGHTH WEEK.—

Conversion of Callus into Bone.—Callus becomes more dense by further calcification which surrounds its vessels. Original bone becomes rarefied, and its vessels become continuous with those of the callus.

FINALLY.—External and internal callus disappear by rarefying osteitis. Intermediate callus becomes condensed into hard bone by sclerosing osteitis, and this change spreads into the ends of the bone. This may take one or two years in adults.

EXCEPTIONAL CASES.—

In Irregular Fractures with much displacement. The angles between fragments are filled up—Projecting corners of bone removed—Architecture of the bone is remodelled.

Comminuted Fractures.—All the fractures are 'set' in callus (*Fig.* 53, B)—Callus becomes densified and old bone rarefied until whole is welded into a homogeneous mass.

When Mobility is not Prevented.—Excessive external callus is formed —Sometimes cartilage occurs instead of bone.

In Many Gunshot Fractures the progress of repair may be very slow, so that after 6 or 8 months there is little firm callus, and this easily bends. This is due to sepsis, and the deprivation of vascular supply to the injured bones.

Flat Bones of Skull.—With no mobility little repair occurs—No callus is formed—Inner table is united most firmly.

Articular Cartilage is replaced by fibrous tissue; rarely by cartilage.

Rib Cartilages are repaired by bony callus

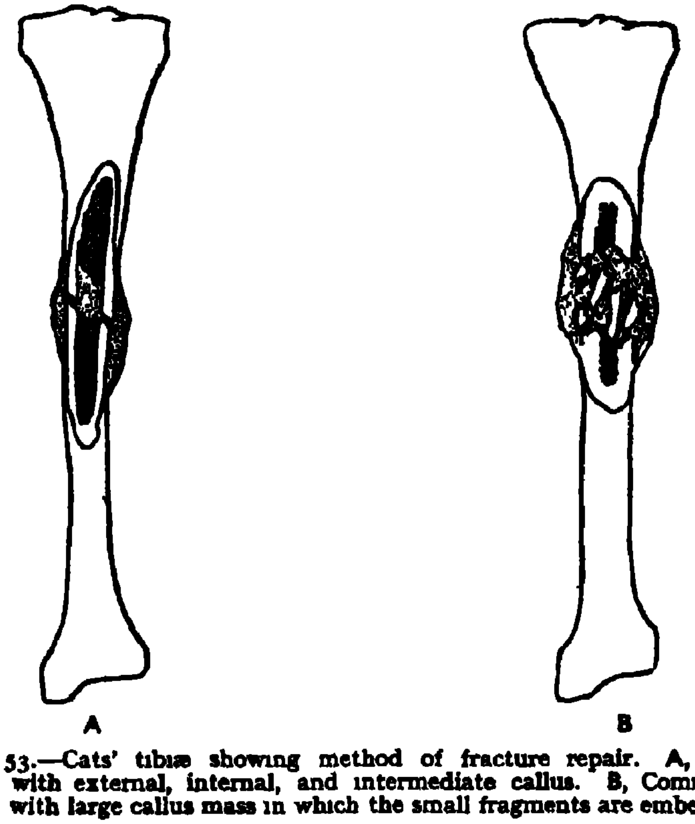

Fig. 53.—Cats' tibiæ showing method of fracture repair. A, Simple fracture, with external, internal, and intermediate callus. B, Comminuted fracture, with large callus mass in which the small fragments are embedded.

Microscopical Changes in the Repair of Fractures.—These are exactly similar to the repair of soft parts, with the addition of the processes of calcification and ossification. Thus:—

BLOOD-CLOT between the fractured ends.

Invasion of clot and replacement by LEUCOCYTES AND FIBROBLASTS.

HYPERPLASIA of tissues with cellular and plastic exudation.

VASCULARIZATION of this cellular layer by new vessels.

DEPOSIT OF LIME SALTS between the vessels.
  Vessels thus lie in tubes of calcareous matter.

CALCAREOUS TUBES are lined by endothelial cells and by layer of osteoblasts, each derived from fibroblasts.

OSTEOBLASTS lay down lamellæ of true bone arranged concentrically round the vessels, some cells running between the lamellæ as Haversian cells.

LARGE FIBROBLASTS CALLED OSTEOCLASTS erode the surfaces of the original bone and make it porous, and enlarge the Haversian canals by the same process.

Thus building up of new bone by osteoblasts in callus, and absorption of old bone by osteoclasts, proceed simultaneously and produce homogeneity.

Temporary callus is absorbed by osteoclasts removing the calcified granulation tissue.

Permanent callus is converted into true bone by osteoblasts.

Condensation of this tissue and of the ends of the bone occurs by continuation of the same process.

Treatment of Simple Fractures.—Four cardinal rules :—

1. PREVENT FURTHER INJURY by 'first aid'.
2. ACCURATE REDUCTION controlled by X rays.
3. EFFICIENT FIXATION of the broken bone, until union is firm.
4. ACTIVE EXERCISE of neighbouring joints and the rest of the body.

REDUCTION OF THE FRACTURE.—

EXTENSION of the limb with effective counter-extension.

MANIPULATION of the broken ends.

CONTINUOUS TRACTION is necessary sometimes to overcome muscular spasm.

A POSITION OF SEMI-FLEXION of the joints so as to relax the muscles.

AN ANÆSTHETIC to abolish muscular spasm in all difficult cases.

COMPLETENESS OF REDUCTION should be estimated by comparative measurement of sound side, and by X rays.

TIME LIMIT FOR REDUCTION.—Within the first week errors of reduction can easily be corrected. After the tenth day parts are 'set' in calcified granulation tissue, and reposition is more difficult.

FIXATION OF FRACTURES—

1. SPLINTS.—Wood, metal, or plaster

If the fracture is in the mid-shaft, the splint should fix the joint above and below.

If the fracture is near a joint, then that joint must be fixed by the splint.

Wood or metal splints must be padded so as not to press too much on bony points—Wide enough to cover the limb—Applied in the position in which the part is to remain.

In severe fractures the limb should always be elevated, so as to prevent swelling of the distal parts.

2. **Plaster Casts.**—The plaster should be put on without any padding, i.e., skin tight. In special positions, e.g., the pelvis, the bony points are protected with felt pads. The main cast is built up with longitudinal slabs, and these are lightly united by circular bandages. The cast should not be put on until danger of swelling is over; alternatively the patient should be kept under observation, and the cast split longitudinally if pressure signs appear. (*Fig.* 54.)
3. **Continuous Traction.**—This may be attached to the limb by adhesive plaster or transfixion pin or wire, applied direct to the distal end of the bone. The traction may be by a weight, cord, and pulley, or by a spring of rubber or steel. Usually the limb is supported by some type of metal or wooden splint.
4. **Double Transfixion Combined with a Plaster Cast.**—In difficult cases, especially the forearm or leg bones, pins or wires are made to transfix both fragments, and a plaster cast is applied so as to incorporate the transfixion pins or wires (*Fig.* 55)
5. **Operation**, with mechanical fixation of the fragments: (*a*) When reduction cannot be effected (*b*) When reduction cannot be maintained—oblique fractures; especially in bones of the leg. (*c*) In some open fractures. (*d*) In fractures with certain complications, e.g., an injury to a main artery, nerve, or joint. (*e*) In transverse fractures of the patella, olecranon, and neck of the femur.

   Advantages.—Accuracy of reduction. Complications can be dealt with. Excessive effusion can be removed. Active movements can be performed earlier; and functional results will be better.

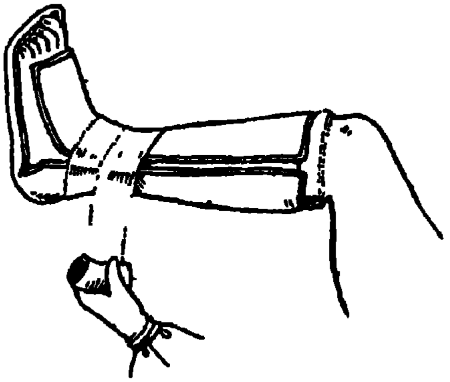

Fig. 54 —Application of plaster 'flats'. Wet gauze bandage applied over flats before circular plaster is used.

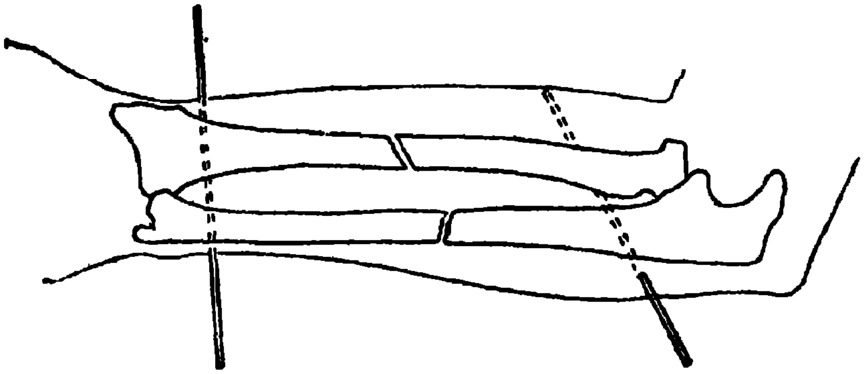

Fig. 55.—Double transfixion of forearm bones, to be incorporated in a plaster cast.

Fixation of Simple Fractures by Operation, *continued.*

Disadvantages.—Sepsis may occur and produce necrosis. Screws or wire may cause pain, irritation, or suppuration, and have to be removed later. The essential tissue-repair is delayed.

Methods.—Simple impaction, i.e., the fitting together of the jagged bone ends. Transfixion of both fragments by a pin projecting through the skin, and incorporated in the plaster cast, which can be removed without taking off the latter. Wire or bands. Vitallium plates fixed by short screws.

After-treatment of Fractures.—

ACTIVE MOVEMENTS.—Early active movements of all joints not actually fixed by retentive apparatus is the one essential for rapid recovery of function. Prolonged fixation of the joints next to the fracture will not lead to permanent stiffness if fixation is efficient and prolonged until union has occurred.

FREEDOM FROM PAIN.—This is the critical test as to efficiency of fixation and the propriety of the movements used. If pain occurs, it shows that the fracture is not properly fixed or that movements are too free.

RESUMPTION OF FUNCTION.—When possible, purposeless active movements should be followed by natural functional movements, e.g., the hand can be used for sewing, the leg for walking, or the spine for bearing weights within one to seven days of the application of a suitable plaster cast.

THE ROLE OF MASSAGE AND PASSIVE MOVEMENTS.—These cannot be applied without removal of the splint. Their benefit is not comparable to that of active muscular contraction. Applied to fractures near the joints, they involve a great danger of displacing the fracture, causing pain and œdema, and it may be myositis ossificans. These dangers will be avoided if their use is postponed until the fracture has firmly united But if active movements and early function have been carried out, they will seldom be required.

Complications arising during the Course of Treatment.—

HYPOSTATIC PNEUMONIA—BEDSORES: In old people confined to bed.

CRUTCH PALSY, generally of the musculospiral.

ISCHÆMIC PARALYSIS, or Volkmann's contracture (p. 158).

GANGRENE, caused by: Immediate injury to the soft parts—Subsequent thrombosis of the vessels—Rupture of the vessels—Septic inflammation in a compound fracture—Bandage applied too tightly—Swelling of a part under a bandage—Flexion of a limb after bandaging.

SLOUGHING, from pressure of a badly-padded splint.

MYOSITIS OSSIFICANS.

Open (Compound) Fractures.—Owe all their special features to :—

SEPTIC INFECTION through the wound. If this does not occur, or can be removed, the case progresses and is treated like a simple fracture.

COMPLICATIONS AND RESULTS IN SEPTIC CASES.—Necrosis of part of the bones—Osteomyelitis.—Septicæmia or pyæmia—Secondary hæmorrhage.

PROCESS OF REPAIR IN SEPTIC CASES.—The inflammatory changes, instead of being plastic, are destructive in character. Pus is produced instead of granulation tissue. Bones in infected area have their vessels thrombosed by septic clot, and hence necrose. Necrosed bone is cast off. Wound and gap between bones fill with granulation. The granulations calcify and then ossify.

Time taken by repair is much longer than in aseptic cases, as this time is required to separate the necrosed sequestra.

Treatment of Open (Compound) Fractures.—

EARLY CASES—within 12 hours of infection.—

Give antitetanic serum—500 units—as prophylactic.

ANTI-SHOCK TREATMENT.—Warmth, hot drinks, a full dose of morphine.

DISINFECTION.—Wound lightly packed with gauze soaked in flavine. The surrounding skin is dry shaved and painted with flavine. (Avoid irrigation or soap and water in and round the wound.)

EXCISION OF WOUND.—A narrow margin (2 mm.) of the skin edge cut away all round. All exposed tissues to have their surface removed for a depth of about 2 mm., the soft parts by clean paring with a razor or knife, the bone by a sharp chisel.

THE FRACTURE.—Can often be manipulated into good position. Never use any internal fixing screws or plates.

Sulphanilamide powder or penicillin is dusted in the wound.

Close the wound by interrupted silkworm stitches.

SPLINTING —A plaster cast with or without traction by transfixion.

LATE CASES—more than 12 hours after infection, or when the above method has failed.—

Complete wound toilette—excision, débridement.

Wound must be made shallow, so that all crevices are opened up.

The wound is insufflated with sulphanilamide powder and lightly packed with vaseline gauze. No sutures are used.

The fracture is brought to full length and alinement by manual or mechanical traction, aided if necessary by transfixion pins placed above and below the lesion.

The whole limb is encased in plaster—pins, wound, and fracture—without any window.

The plaster case is left alone without any cutting for 4 to 8 weeks, in spite of the disagreeable odour. Only if pain and persistent temperature occur is the plaster cut open. (Winnett Orr.) Or a window may be cut over wound and pack changed weekly.

Alternative Method —The wound may be left open, a window being cut from the plaster cast over it. (Böhler.)

Trueta uses sterile gauze to pack the wound, followed by a plaster cast. This has proved of great value in war wounds.

Non-union of Fractures may be of three types: (1) Absolute non-union; (2) Fibrous union; (3) False joint.

COMMONEST SITUATIONS —Bony processes giving muscular attachments (patella, olecranon, coracoid process, os calcis)—Humerus—Femur (especially the neck)—Tibia.

CAUSES.—

WIDE SEPARATION OF THE FRAGMENTS (e.g., olecranon).

LOSS OF SUBSTANCE (e.g., in gunshot fractures).

CONSTANT MOVEMENT (e.g., humerus).

INTERPOSITION OF SOFT PARTS (e.g., patella).

DEFECTIVE BLOOD-SUPPLY (e.g., head of femur).

GENERAL BONE DISEASE: Osteomalacia.

Non-union of Fractures—Causes, ***continued.***

Local Bone Disease: Malignant tumour—Gumma—Tubercle.

Scurvy or General Asthenia: Very rare.

N.B.—In senility, rickets, fragilitas, paralysis, tabes, bones break easily but heal readily.

In Children and Young Adults the cause is very obscure—possibly an undue formation of cartilage instead of bone from the callus.

PATHOLOGY.—There are two types of non-union in the long bones.

The Atrophic Type—This is usually the result of loss of substance with want of contact between the bone ends. The fragments become spindle-shaped and very thin and fragile owing to the absorption of bone salts. The medullary cavity is enlarged and filled with fat.

The Hypertrophic Type. Pseudo-arthrosis.—The bone-ends become thickened and densely sclerosed. The medullary cavity becomes obliterated by dense bone for some distance from each end. The end margins produce osteophytes like candle guttering. A fibrous capsule with synovial fluid is formed between the ends. The whole resembles an osteo-arthritic joint.

SIGNS OF NON-UNION—Unnatural mobility—Pain: especially in fibrous union.

TREATMENT.—

Non-operative—Passive congestion. Active use of limb, supported by suitable splint, e g, a plaster or leather case. Improvement of general health

Subcutaneous Drilling (Beck's method).—The end of each fragment is drilled by a fine drill like a Kirschner's wire, in 6 to 12 channels (*Fig.* 56).

Operative Vitalization.—Excision of intermediate scar tissue. Cutting fresh surfaces to bone ends

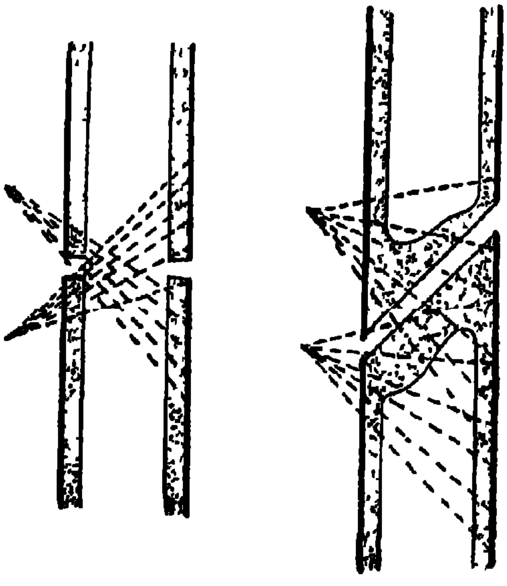

Fig. 56.—Diagram of transverse and oblique fractures showing the lines of multiple drill holes

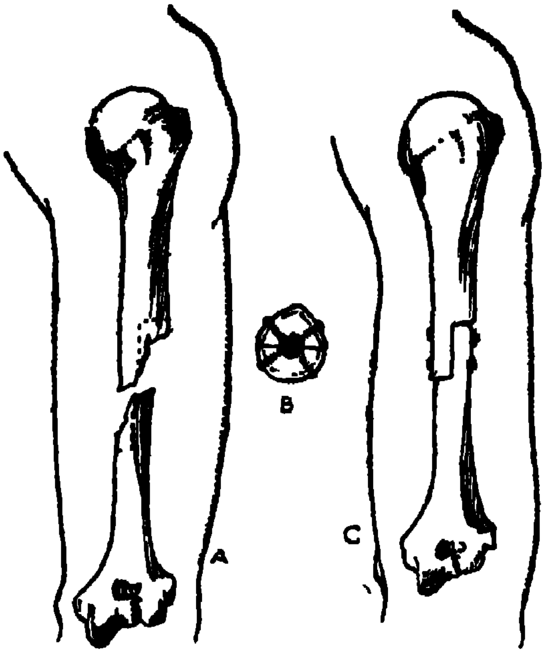

Fig. 57.—Step-cut method of uniting a fracture of the humerus Suitable for ununited fractures A, Before operation—dotted lines show lines on which the bone is cut; B, Transverse section of union, showing pins or bolts in different radii, C, Step-cut union, providing a large Z-shaped area of contact between the fragments

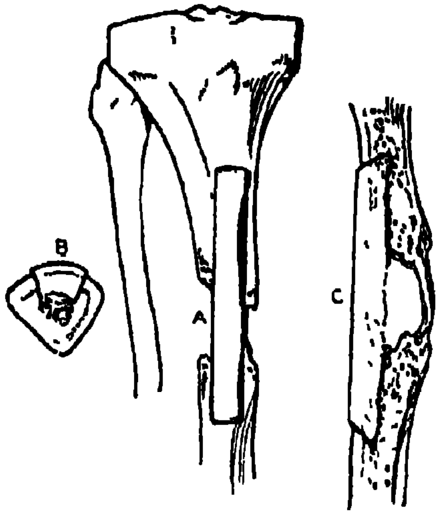

Fig. 58.—Cortical or 'inlay' method of bone-grafting. A, Shows graft lying in place from the front; B, Cross-section showing wedge-shaped graft from opposite tibia lying in place; C, Longitudinal section through graft and host bone showing method of shaping the ends of the graft.

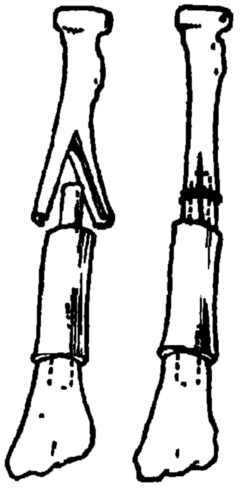

Fig. 59.—Intramedullary method of fixing graft. The graft is shaped like a cricket-bail. One end is driven into the distal fragment, the other is inserted into the proximal fragment by splitting the latter and then tying it over the graft.

Non-union of Fractures—Treatment, ***continued.***

OPERATIVE FIXATION.—

1. *Step-cut Operation.*—Shape each bone end by cutting out a step; fit the ends together and fix by suture, bone nails, or both. This is specially suited for the humerus, where the loss of length entailed does not signify (*Fig* 57).
2. *Cortical Bone-graft.*—A long groove is cut in both fragments. Into this is fitted a bone-graft cut from the tibia. Specially suited for the femur or tibia (*Fig.* 58).
3. *Medullary Bone-graft*—The ragged ends of the fracture are cut off. A graft is cut from the crest of the tibia and its ends are shaped as pegs. The peg ends of the graft are fitted into the marrow cavity of the ends of the broken bone. Specially suitable for gap-fractures of the radius (*Figs.* 59, 63).
4. *Massive Bone-graft.*—This is a variety of 2 and 3. Each fragment has about one-third of its wall cut out, leaving a deep groove into the marrow cavity. A stout bone-graft is laid in this and tied in place by wires, kangaroo tendons, or vitallium screw, or the graft may be driven into the bored-out marrow cavity of one fragment and laid with a cortical groove in the other (*Fig.* 60).

N.B—It is absolutely essential, after all these methods, to fix the limb in suitable splint or plaster for a period of not less than 3 to 6 months in the arm and 6 to 12 months in the leg.

FRACTURE OF SPECIAL BONES

INFERIOR MAXILLA

CAUSES.—Direct violence (common), indirect (rare).

POSITION OF FRACTURE.—Between canine tooth and mental foramen—Behind angle of jaw—Coronoid process (rare)—Condyle (rare).

SIGNS.—Crepitus—Irregularity of line of teeth—Wound and bleeding into mouth—Always compound owing to communication with mouth—

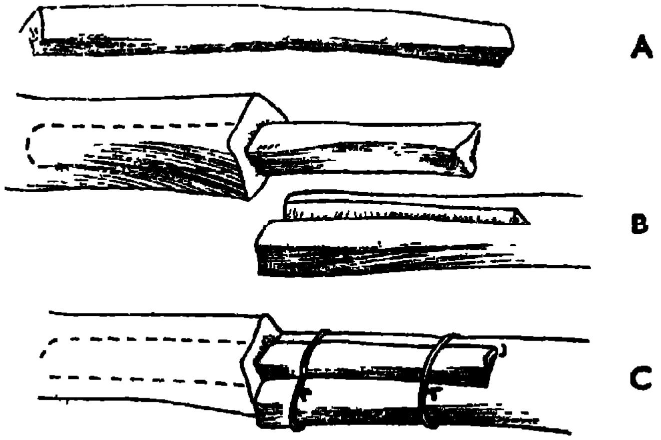

Fig. 60.—Massive bone-graft for tibia or femur. A, Graft cut from opposite tibia; B, Graft driven into marrow cavity of upper fragment, whilst a slot is cut for it in the lower; C, Graft in position.

Displacement: anterior fragment downward, posterior fragment upward. —Pain due to involvement of inferior dental nerve.

OF CONDYLE: This process is drawn forwards or inwards.

OF CORONOID PROCESS: It is drawn upwards.

COMPLICATIONS.—In compound cases: Necrosis—Septic pneumonia —Pyæmia.

TREATMENT.—

1. Barrel bandage described by Gillies and Fry.
2. Gunning's interdental splint, of non-corrosive metal, fitting over crowns of teeth and fixed by metal bars to a similar splint in the upper jaw.
3. Hammond's wire splint round the teeth.
4. Bone-grafting in cases of loss of substance due to gunshot wounds. The graft is best taken from the crest of the ilium.

Immobilization is required for three weeks. Feed patient between cheek and teeth. Use frequent antiseptic mouth-washes.

(For fractures of the nasal bones, *see* Chap. XXVIII)

Fracture of the Neck of the Condyle.—This is often missed. Great care in manipulative reduction is required, if this fails an open operation should be done. Impaction over a peg. In late cases the condyle must be removed.

RIBS

CAUSE.—Indirect violence (commonest); ribs break near the angle. Direct violence

RIBS AFFECTED.—Fifth and eighth most commonly.

SYMPTOMS.—Pain on breathing—Irregularity and crepitus—Pain over fracture on pressing sternum to spine.

COMPLICATIONS (especially in cases of direct violence).—Pleurisy—Pleural effusion: serous, purulent, blood, or air—Wound of lung: hæmoptysis—Surgical emphysema

TREATMENT.—Strapping completely round the chest; applied in position of expiration. Firm bandage.

When thoracic complication exists—Rest in bed

CLAVICLE

CAUSES.—Indirect violence by falls on hand—Direct violence (rare).

FOUR POSITIONS:—

1. STERNAL END (rare): Little displacement—Inner end drawn up.
2. MIDDLE (common situation): Between rhomboid and coraco-clavicular ligaments—Fracture runs backwards and inwards—Inner end raised—Outer end depressed, tilted down, approximated to mid-line—Head bent over to affected side—Shoulder displaced downwards, inwards, and forwards.
3. BETWEEN CONOID AND TRAPEZOID LIGAMENTS (direct violence): Slight displacement—Contusion and crepitus.
4. AT ACROMIAL END (direct violence): Outer end downwards and forwards.

VARIETIES AND COMPLICATIONS.—Greenstick fracture (common in children)—Compound (rare)—Injury to subclavian vein (rare)—Injury and thrombosis of subclavian artery: gangrene—Wound of pleura and lung.

Clavicle, *continued.*

TREATMENT.—

REPLACEMENT.—Pull shoulder backwards and upwards. Manipulate fragments.

RETENTION.—Required for three weeks.

1. REST IN BED.—Pillow between scapulæ. Arm bandaged to side. Callus formation reduced to a minimum. Hence useful for ladies.
2. BANDAGING both shoulders back by a figure-of-8 bandage. Supporting arm in a sling. With patient (recumbent at first) all the joints can be actively exercised daily.

PROGNOSIS.—Usually the patient can return to work in six to eight weeks.

SCAPULA

VARIETIES.—

ACROMION.	Result from direct violence. Cause slight deformity or crepitus. Treat by raising and fixing the arm.
CORACOID PROCESS	
BODY	

NECK.—

Anatomical neck: Rare complication of a dislocation.

Surgical neck: Fracture runs downwards from the supra-scapular notch. The separated piece includes the coracoid process.

Signs.—Flattened shoulder—Prominent acromion—Lengthened arm.

Treatment.—Axillary pad—Elevate shoulder—U-shaped leather splint for shoulder.

PROGNOSIS.—This is usually bad, owing to traumatic neurasthenia, osteo-arthritis, or adhesions about the joint Eight weeks to a year may be taken as the limit of time before which the patient can return to work.

HUMERUS

Upper End.—

1. NECK OF HUMERUS.—

VARIETIES.—

a. Without displacement. A contusion and fracture of neck of humerus,

b. With adduction of distal fragment

c. With abduction of distal fragment.

CAUSES.—Contusion and fracture are due to a direct blow on the outer aspect of the shoulder, types (*b*) and (*c*) due to a fall on the outstretched hand.

SIGNS.—Loss of mobility, marked bruising of tissues, pain on movement, occasionally shortening of limb.

TREATMENT —

a. Contusion and Fracture.—Invariably subperiosteal injury without displacement. Unnecessary to immobilize limb. Early exercises.

b. Adduction Fracture.—Outward angulation with impaction on the inner side and the shaft of the humerus adducted. If not corrected, abduction limited. In old patients (over 50) impaction not broken down, active movements begin at once. Younger people—correct deformity by traction and abduction of limb in an abduction frame for 4 weeks.

Adduction fracture in children—similar deformity to the above, and if fracture near the epiphysial line, is known as a displacement of the humeral epiphysis. If epiphysial line damaged, unequal growth of the arm may occur.

c. Abduction Fracture.—Displacement opposite to the above, always associated with a fracture of the great tuberosity if there is impaction. Treated by supporting in a sling and early movement; do not immobilize or treat in abduction frame.

2. GREAT TUBEROSITY.—

CAUSES.—Direct violence or indirect, as fall on outstretched hand. Fracture line usually through surgical neck, occasionally through anatomical neck, in young people through the diaphysial side of the epiphysial cartilage (separated epiphysis).

DISPLACEMENT.—Fracture is transverse or oblique without displacement, or with medial (adduction type) or lateral (abduction type) displacement of the lower fragment. Impaction may occur.

SIGNS.—Bruising and widening of the shoulder—Marked crepitus.

TREATMENT.—Fixing the arm on an abduction splint, if separation of fragments.

Open operation may be indicated in some cases, especially young patients, or those in whom there has been also a dislocation of the shoulder. The upper part of the deltoid is split, and the tuberosity accurately nailed to the head by a steel or ivory nail.

Shaft.—

CAUSES.—Direct, indirect, or muscular violence.

DISPLACEMENT.—

1. Below the pectoral insertion and above the deltoid: Upper fragment inward, lower fragment upwards and outwards.
2. Below the deltoid insertion: Upper fragment outwards, lower inwards and upwards.
3. In lower third: Lower fragment upwards in front of or behind the upper.

COMPLICATIONS.—

Injury of the musculospiral nerve or its involvement in callus.

Non-union, due to: Interposition of fibres of the triceps muscle or defective immobilization.

TREATMENT.—

1. In easy cases, a plaster U-shaped slab from the axilla on inner side of arm, under the elbow and then up to the shoulder on outer side. Union occurs quickly in the spiral type of fracture.
2. In difficult cases, in addition to the above, a posterior slab from the shoulder to the knuckles. The whole arm is placed on an abduction splint with traction. (*Fig.* 61.) This applies to the transverse type of fracture; union is slow, accurate immobilization is essential. A plaster shoulder spica is admirable.
3. Open operation. Only required in fracture of the mid shaft with involvement of the musculospiral nerve. The nerve may be found caught between the fragments. The nerve should be freed, and, if cut, should be sutured. The bone is joined by a plate or peg (*Fig.* 62).

Lower End.—

1. TRANSVERSE SUPRACONDYLOID.—

CAUSES.—Indirect violence by falling on the hand with a bent elbow Direct violence by falling on the bent elbow.

Humerus—Lower End—Transverse Supracondyloid, *continued.*

DISPLACEMENT.—Lower fragment upwards, usually behind, rarely in front of, the upper.

SIGNS.—Shortening of the arm from acromion to condyle. Increase in thickness of the lower end of the humerus from before backwards. Unaltered relation of condyles to olecranon. Apparent shortening of the forearm according to backward displacement.

TREATMENT.—

Fix in position of flexion after manual reduction, by slinging the wrist to the neck, so that the hand lies on the opposite shoulder, using a posterior plaster slab. It is important to correct any lateral displacement as well. The degree of flexion should never be such as to interfere with the circulation, as judged by the radial pulse.

If accurate reposition is not possible, there are two alternatives, viz.: (1) Apply skeletal traction by a pin or wire through the olecranon, the hand being hung to a Balkan beam, the elbow being at a right angle; (2) Open operation through a posterior incision, impaction of the fragments in correct position, with the aid of kangaroo-tendon sutures if necessary.

COMPLICATIONS.—Myositis fibrosa (Volkmann's ischæmic contracture) and myositis ossificans.

2. SEPARATION OF THE LOWER EPIPHYSIS.—May occur up to fourteen or fifteen. Common in children. After fourteen or fifteen it does not involve the internal condyle. Displacement is backwards and outwards.

TREATMENT.—Reduce by flexion Fix in position of flexion by splints or bandage. Sling for the forearm Fixation for three weeks.

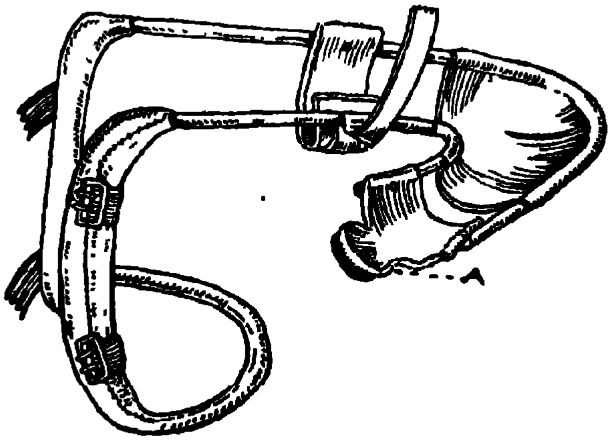

Fig. 61.—Abduction shoulder splint. A, Adjustable cock-up for wrist.

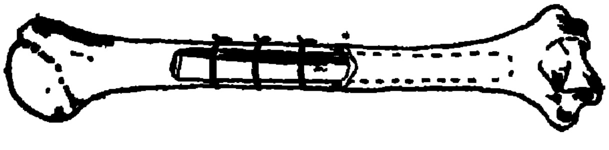

Fig. 62.—Bone-graft of humerus.

3. CONDYLES.—
   INTERNAL CONDYLE.—Direct violence:—
   - *a.* Intracapsular: Involves the epicondyle and part of the trochlea. May result in cubitus varus.
   - *b.* Extracapsular: Involves the epicondyle only.

   EXTERNAL CONDYLE.—Always intracapsular. Involves the epicondyle and part of the capitellum May result in cubitus valgus, with the late complication of delayed ulnar neuritis. The fragment may be rotated, so that the articular surface looks outwards

   SIGNS.—Great swelling and ecchymosis of the joint. Crepitus on grasping the affected condyle. The extended elbow can be adducted or abducted.

   TREATMENT.—If displacement is easily reduced, sling the arm with the elbow flexed If X rays show that reduction is from the first unsatisfactory, then open the part and fix the condyle by peg or wire.
4. T-SHAPED FRACTURE INTO THE JOINT.—Rapid effusion of blood into the joint. Crepitus on moving the condyles on one another.
   TREATMENT.—As above. Open operation or skeletal traction through the olecranon is indicated if reduction cannot be effected.

Prognosis after Fractures of the Humerus.—

THE BEST RESULTS follow fractures of the shaft without complications Work to be resumed in two or three months

BAD FUNCTIONAL RESULTS may be expected in.—

FRACTURES INVOLVING THE SHOULDER OR ELBOW, especially the former. Permanent disability is often produced in labouring men. Six months may be regarded as the shortest period of convalescence.

FRACTURES TREATED BY LONG FIXATION WITHOUT ACTIVE MOVEMENTS. —In cases near the joints permanent disability results

INJURY TO THE BRACHIAL PLEXUS usually causes permanent inability for hard work

INJURY TO THE CIRCUMFLEX OR MUSCULOSPIRAL NERVE —In favourable cases one or two years elapse before work can be done

ULNA

Olecranon.—

CAUSES.—Falls on the elbow, or muscular action (rare)

DISPLACEMENT —Drawn upwards and backwards by the triceps.

SIGNS.—Mobility, with crepitus of the fragment Great distension of the joint

COMPLICATIONS —Forward dislocation of the forearm at the elbow. Injury of the ulnar nerve Fibrous union

TREATMENT.—
1. Open operation and wiring Active movements after one week.
2. Excision of the olecranon and suture of the triceps expansion.
3. If an operation is refused, straight posterior splint for 6 weeks.

Coronoid.—Occurs as a complication of backward dislocation of elbow. The dislocation is easily reduced, is accompanied by crepitus, and easily recurs.

TREATMENT.—Fixing in full flexion. Elbow should not be straightened for three to four weeks, but all the other joints should be actively exercised.

Ulna, *continued*

Shaft.—

CAUSE.—Direct violence

COMPLICATION.—Dislocation of the radius—i e., Monteggia fracture either of the flexion or extension type.

DISPLACEMENT.—Lower fragment towards the radius (pronator quadratus), upper fragment forwards (brachialis anticus)

TREATMENT —As in fractures of both bones.

Styloid Process.—May complicate Colles's fracture of the radius.

RADIUS

Often associated with injury to other bones forming the elbow-joint

Head and Neck.—

VARIETIES —

1. Head may be impacted into the neck
2. Vertical fracture of the outer third of the head, with downward displacement, i e, marginal fracture
3. Fragmentation of the head

TREATMENT —

1. Manipulation, with forearm in full supination, exerting forcible pressure on the broken fragment, and plaster cast from mid-arm to metacarpal heads in full supination
2. Excision of head and neck through posterior incision Must be done early

Shaft.—

1 ABOVE PRONATOR TERES INSERTION.—

Lower fragment fully pronated and drawn inwards (both pronators)

Upper fragment fully supinated (biceps and supinator brevis).

TREATMENT —Manipulation and plaster, as for head and neck, *above*

2. BELOW PRONATOR TERES INSERTION —

Upper fragment drawn forwards and inwards (biceps and pronator) in position between supination (biceps) and pronation (pronator)

Lower fragment inwards by the pronator quadratus

TREATMENT.—As above, but the hand is placed midway between pronation and supination.

OPERATIVE TREATMENT OF FRACTURED RADIUS.—In all cases where displacement is not well reduced by manipulation, immediate operation should be done by pegging (*Fig.* 63) or plating.

Lower End, Backward Displacement (Colles's Fracture).—

CAUSES.—Falls on the outstretched palm. Common in elderly women.

POSITION.—Within one inch of the joint

DIRECTION.—Transverse, or, more commonly, oblique from above downwards, forwards, and inwards.

DISPLACEMENT.—Is of a quadruple nature. The lower fragment is displaced:—

1. Upwards towards the elbow.
2. Backwards by the direction of the blow and direction of the limb.

3. Rotated round the ulnar styloid as a centre, so that the outer part is more displaced than the inner.
4. Rotated backwards round the line of fracture, so that the back of the fragment is more displaced than the front.

DEFORMITY.—Is also of a fourfold character.—

1. Radial styloid is higher than normal, i.e., it is on a level with the ulnar styloid instead of being below it.
2. Hand is displaced backwards, and there is a bony prominence at the back of the wrist, with another bony prominence at a higher level in front of the wrist (dinner-fork deformity)
3. Hand is abducted to the radial side, with marked prominence of the ulnar styloid process
4. Wrist is rotated backwards, so that the joint surface looks backwards as well as downwards.

IMPACTION is usually well marked. Hence crepitus and mobility will be absent and the fracture may be mistaken for a sprain. The relative position of the styloid processes is the most important point in guarding against this.

The posterior part of the shaft is driven into the lower fragment.

The anterior part of the fragment is driven into the shaft (*Fig.* 64).

TREATMENT.—Early reduction of the deformity is absolutely essential, preferably within six hours

Anæsthetic: General or local In the latter 2 per cent novocain is injected into the region of the fracture; it has the advantage that if X rays show reduction incomplete, fresh manipulation can be done.

Reduction by pulling the hand, pressure on the lower radial fragment, or bending the hand backwards so as to increase the deformity; once disimpaction occurs reduction is easy

Four manœuvres are required in reduction—traction, ulnar flexion, ulnar deviation, and pronation

Plaster Splint. A slab of plaster is moulded on the back of the hand and forearm, reaching from the knuckles to the elbow, and wide enough to

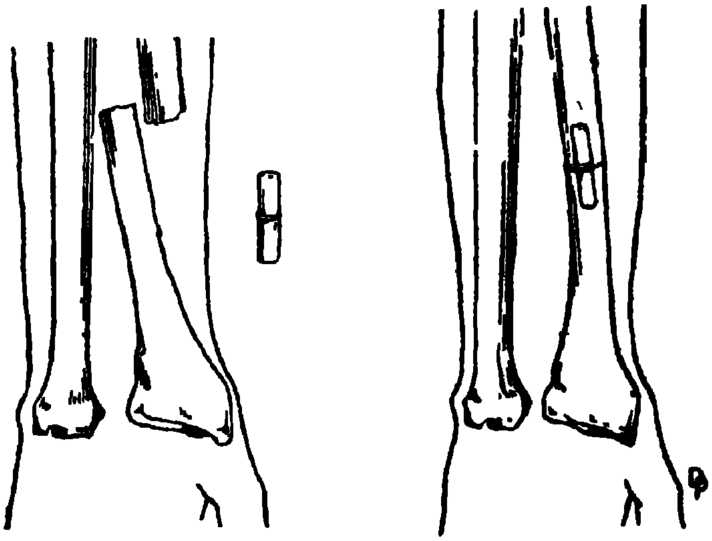

Fig. 63.—Treatment of fractured radius by intramedullary peg.

Radius—Colles's Fracture—Treatment, *continued*

embrace both sides of the wrist. It is fixed by a wet muslin bandage or a circular plaster bandage, but if the latter is used it must not come below the middle palmar skin crease (*Fig.* 65).

Movements Fingers, thumb, elbow, and shoulder regularly and actively moved every day Purposive work begun within the first week.

Plaster removed at the end of three weeks to one month, followed by systematic regular active exercises

'Chauffeur's Fracture'.—This is caused by the back-firing of a motor engine whilst the chauffeur is starting it with a handle. The outer part of the lower end of the radius, with the styloid process, is cracked off but not much displaced The symptoms are those of a severe sprain. Treatment as in Colles's fracture

Lower End, Forward Displacement (Smith's Fracture).—

CAUSED by falls on the back of the flexed wrist

Similar to a Colles's, but all the posterior displacements are now anterior. Produces a rather more square angle, which has given it the name of 'the gardener's fork'

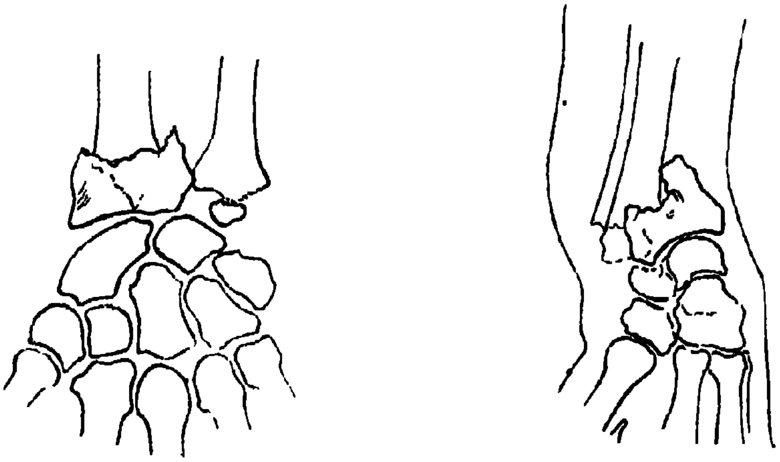

Fig 64 —Colles's fracture—antero-posterior and lateral X-ray views

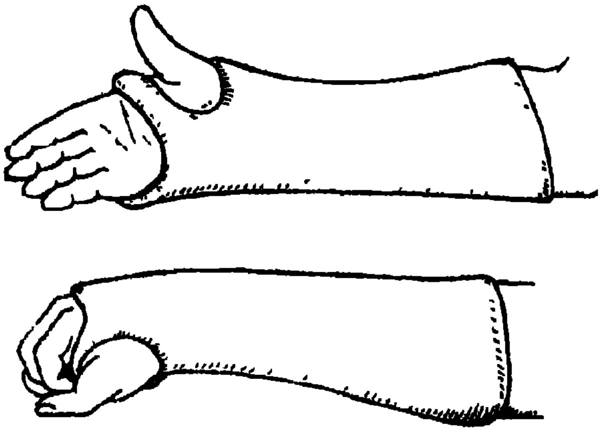

Fig. 65 —Colles's fracture in plaster case.

Separated Lower Epiphysis.—Occurs up to twenty.

Similar to a Colles's fracture. The displacement is more marked Impaction is rare.

Treatment is the same.

Both Ulna and Radius.—

VARIETIES.—

Direct violence: Both bones broken at seat of impact

Indirect violence, falls on hand: Ulna broken at a different level from radius.

DISPLACEMENT.—Broken ends tend to be drawn together by the pronators and supinators

TREATMENT.—Reduction may be easy or very difficult. It is effected by flexing the elbow to a right angle, counter-traction to the arm above the elbow by a wide band tied to a fixed point, and steady pulling on the hand.

1. If reduction is effected easily: Put up in plaster cast for four to six weeks. Cast is from the deltoid insertion to the knuckles. It consists mainly of long anterior and posterior slabs. Fingers, thumb, and shoulder exercised actively every day.
2. If reduction is difficult, or if displacement recurs. Traction and counter-traction as before Transfixion wires through the upper end of shaft of ulna and through the bases of the 2nd to the 5th metacarpals or lower ends of radius and ulna Plaster cast incorporates these wires (*see Fig* 55)
3. Open operation for cases where closed reduction has failed to produce good position The accurate restoration of alinement of the radius with restoration of its normal curve is essential for skilled workers. The actual operation may be merely impaction after clearing soft parts, e g., the pronator teres tendon from between the ends, or by pegging or plating or bone-grafting If a plate is put on the lateral aspect of the radius it should be suitably curved so as to restore the natural bend of the bone

Note—Synostosis of the shafts never occurs except in septic, open, and especially gunshot fractures

Prognosis after Fractures of the Forearm.—

BAD RESULTS ARE OBTAINED and function often never regained in —

Separation of the coronoid process

Fractures of the neck of the radius or the shaft above the pronator insertion, if displacement has not been corrected.

Colles's fracture, not replaced, or treated by forced passive movements before union is firm.

PROGNOSIS IS DOUBTFUL, and work can only be resumed within three to six months in. Fractures of both bones or Colles's fracture if reduction has not been perfect.

PROGNOSIS IS GOOD in fractures of one bone only, or of both bones if perfect reduction and proper fixation have been effected

BONES OF THE HAND

Caused by direct or indirect injury Of these the most important are those of the scaphoid, first metacarpal, and phalanges.

SCAPHOID.—Often undetected X rays which do not show it directly after the injury will reveal it later on (*Fig* 66) If untreated there is great tendency to non-union.

Bones of the Hand—Scaphoid, ***continued.***

Treatment —Plaster cast for six weeks or more. For late untreated cases: plaster for six months; multiple drilling; bone-graft; or excision.

FIRST METACARPAL —A fracture at the base of the bone which may be impacted.

Treatment —Local anæsthetic. Reduction Put up in plaster splint, including lower end of forearm and thumb

PHALANGES.—

Treatment.—Reduce by traction. Plaster round wrist and up to the back of the knuckles. Finger bent over a wire splint applied to palmar aspect. Tip of finger transfixed by wire through pulp. Wire tied to the end of wire splint and to the wrist-piece (*Fig* 67)

PELVIS

Above the Brim of the True Pelvis.—Parts of the iliac crest or the crest of the pubis (rare) may be broken off by direct violence

TREATMENT —By rest in bed, with pelvic bandage

Involving the True Pelvic Cavity.—

CAUSES.—Crushes or being run over

POSITION —Through the pubic and ischial rami into the obturator foramen. Also secondarily through the sacro-iliac joint of the opposite or same side

COMPLICATIONS —Laceration of the urethra, bladder, vagina, or rectum

TREATMENT.—Rest in bed for six or eight weeks Poroplastic pelvic belt Appropriate treatment for visceral complications. Tie in a catheter for some days if there is hæmaturia or retention of urine

Acetabulum.—

POSTERIOR AND UPPER LIPS, with dorsal dislocation of hip.

Reduce and treat by extension and extreme abduction to prevent recurrence

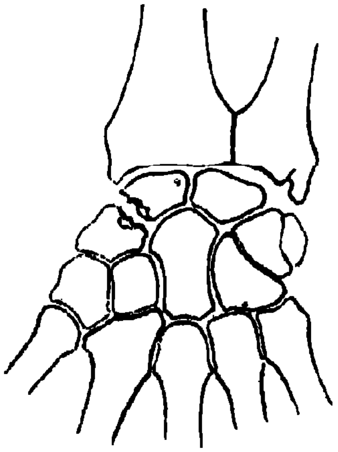

Fig. 66.—Fracture of scaphoid.

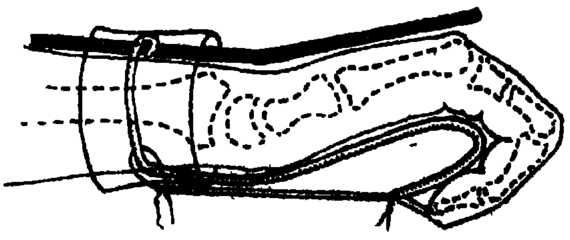

Fig 67.—Traction in fracture of 1st phalanx.

FLOOR OF THE ACETABULUM, the femoral head being driven into the pelvis. Head of the femur can be felt per rectum.
REDUCE under an anæsthetic if possible.

Tuber Ischii, Sacrum, Coccyx.—These fractures require no special description. A fractured coccyx may lead to great pain, or unite in a position in which it encroaches on the pelvis; in either case it is best excised.

FEMUR

Neck of Femur.—May be the high intracapsular or the low extracapsular. These should be subdivided as follows, from the point of view of successful treatment (Watson Jones) (1) Abduction fractures: Impacted subcapital. (2) Adduction fractures (*a*) Subcapital, (*b*) Transcervical, (*c*) Inter- and per-trochanteric.

Abduction fractures produce a fracture line which is almost horizontal, and therefore the shearing strain between the fragments is minimal. Adduction fractures produce a more vertical fracture line. If the angle is 30° then the fracture will unite even without treatment; if the angle is between 50° and 90°, as in adduction fractures, then some form of internal fixation is needed

CAUSES.—Slight violence of an indirect nature. Occurs at any age, but is typical in old people Atrophy of the bony neck, and a more horizontal position of the neck than normal, predispose to its occurrence.

DISPLACEMENT —Limb rotated outwards by its weight and by external rotators. Drawn up by hamstrings and quadriceps, but the capsule prevents much upward displacement The actual shortening is therefore slight—about one inch. The external rotation is not so marked when the fracture line is intracapsular, as the capsular attachments to the distal fragment prevent some external rotation.

REPAIR.—Usually defective. Non-union or fibrous union, due to (1) Bad vascular supply of the head; (2) Difficulty in fixing the fragments in apposition; (3) Interposition of fibres of the capsule.

TREATMENT.—

Depends on the age and the general condition of the patient Feeble people in poor health are best treated by 10–12 lb. extension to the limb and made to move and sit up in bed as soon as possible If, on account of condition of patient, no treatment is to be given, provide with a walking calliper and allow to walk early Age alone is no contra-indication to operative treatment

1 BY PLASTER SPICA according to the technique of Whitman Reduce the fracture by Leadbetter's method of traction on the hip flexed to a right angle, abduction, internal rotation, and then extension. Plaster applied in abduction and internal rotation with the hip extended, from the nipple line to the toes (*Fig.* 68). Plaster retained for 3 months and then reapplied for 2–3 months, according to state of union of fracture

Treatment is: (*a*) Cumbersome; (*b*) Trying for the patient, (*c*) Stiffness of the knee may occur in the aged from prolonged immobilization; and (*d*) It is difficult to control the fragments.

Bony union occurs in 40 to 50 per cent of cases

2 BY INTERNAL FIXATION OF THE FRAGMENTS —Many methods Most used is the three-flanged stainless steel pin modified from

Femur—Neck—Treatment, *continued.*

Smith-Petersen's original nail. Union occurs in 70 per cent of cases The nail (*Fig* 69) is 3 to 4 in. long with three thin steel flanges set an an angle of 120° to the central axis. The axis is hollow for a guide wire (Sven Johansson). The shape of the nail secures very firm fixation when it is driven through the neck into the head. This fixation is so firm that no splinting is required after the operation, and the patient can begin active movements of the hip at once and sit up in bed. Weight bearing after three months

Methods of Insertion—(*a*) Open operation. Only suitable for vigorous patients (*b*) Use of a number of wire guides driven into the neck, choice of one, after X-ray examination, which is in the true axis, driving nail over the selected guide. This usually takes longer than the technique described below. (*c*) The use of a mechanical device for placing the wire guide (*Fig* 70) Many devices are employed, that of Engel-May, Austen-Moore, or Hey Groves may be used The patient is fixed in bed and the fracture adjusted by traction and countertraction Position verified by X rays in two planes. The patient remains in bed for insertion of nail This method is quick and simple, it can be done under gas and oxygen anæsthesia and the actual operation only takes ten minutes. It is therefore the method of choice for elderly and

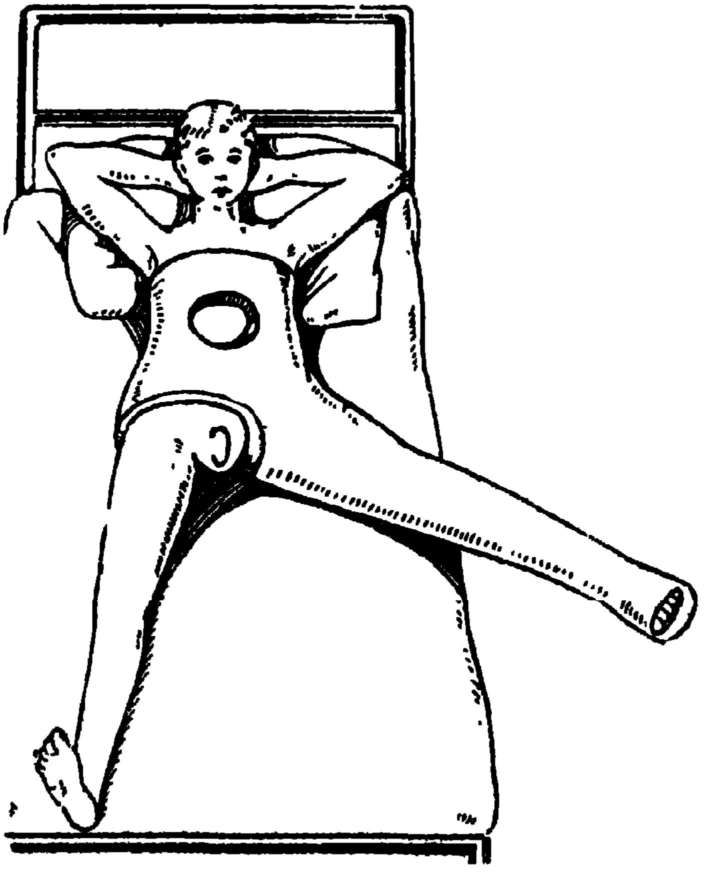

Fig. 68.—Plaster-of-Paris spica for fractured femur (*Whitman*).

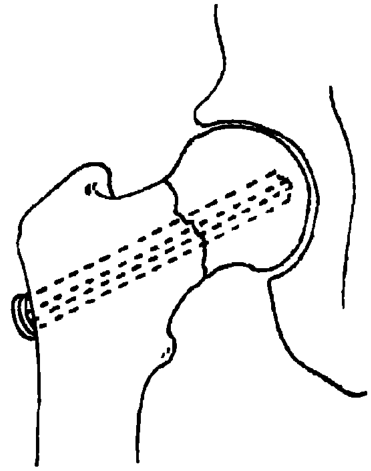

Fig. 69.—The Smith-Petersen pin.

feeble patients. It is important to use the correct length of nail, to insert it correctly so that it is in good position as seen in lateral and anteroposterior X-rays, and it is essential to impact the two fragments after the nail is inserted.

After-treatment.—Free movement in bed; gets up after four weeks. Weight bearing in three to six months. No splint should be necessary. A walking calliper splint is only used if operation has been refused or if it has failed.

Complications of Nailing Operations—

i. Early: (*a*) Inaccurate insertion of the nail. (*b*) Nail too long or too short. (*c*) Inaccurate reduction of the fracture. (*d*) Insertion of guide wire into acetabulum or pelvis.

ii Late: (*a*) Loosening and extrusion of nail. (Prevented by using vitallium nail) (*b*) Fracture of the nail due to imperfections in the metal. (*c*) Avascular necrosis of the head of the femur. More common if any operation has been performed damaging the capsule of the hip-joint.

3. By Osteotomy.—For cases of permanent non-union. (*a*) Lorenz bifurcation: The shaft of the femur engages under the lower margin of the acetabulum (*Fig.* 71). (*b*) Schanz's cuneiform: A wedge is removed from below the trochanters. Two steel pins are inserted above and below the gap, at a diverging angle They are brought into a parallel position and incorporated in the plaster case (*Fig.* 72).
4. By Whitman's Operation —For cases of permanent non-union, or where the head of the femur has undergone necrosis Head is removed, trochanter transplanted into acetabulum.

At the Junction of the Neck with the Trochanters.—

Basal fractures ('extracapsular fracture')

CAUSES.—Great violence directly applied to the trochanter major.

ANATOMY —Cleavage is chiefly in the intertrochanteric line. Comminution is common The neck, great trochanter, and shaft are the chief fragments Impaction is the rule; the neck is driven into the great trochanter. The joint is always involved in front, though the fracture is usually 'extracapsular' behind

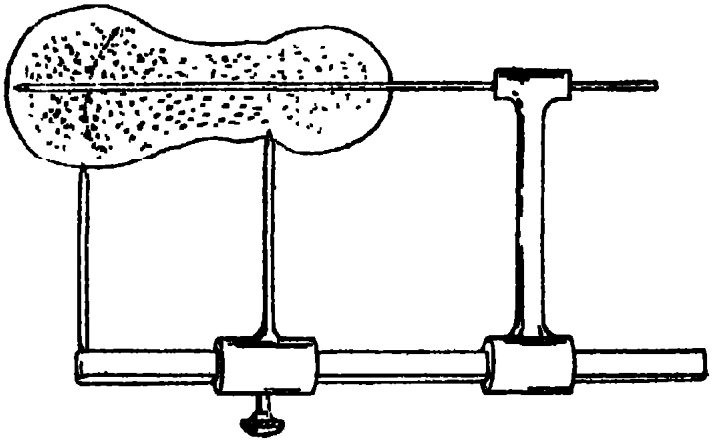

Fig. 70.—Drill guide for fracture of the neck of the femur (closed operation).

Femur—At the Junction of Neck with the Trochanters, *continued*.

DISPLACEMENT.—Leg is rotated outwards by its own weight and the predominance of the external rotators.

Shortening is well marked, and may be two inches The length from the anterior superior iliac spine to the malleolus is shortened. The trochanter is raised above Nélaton's line. The horizontal side of Bryant's triangle is shortened.

The trochanter is often nearer to the mid-line than is normal—the 'bi-trochanteric test'

OTHER SIGNS.—Crepitus is absent when impaction exists. Marked bruising about the trochanter The trochanteric region is thickened. The ilio-tibial band is relaxed The trochanter rotates round the arc of a smaller circle than is normal (if not impacted).

TREATMENT—Always disimpact unless the patient is old and very feeble. Disimpaction requires continuous traction on a Thomas splint. After disimpaction, treatment should be:—

1. Skeletal Traction in a position of abduction for six weeks. Followed by walking calliper for some months
2. Roger Andrews' Well-leg Traction
3. Smith-Petersen's Nail—Is only suitable for cases without comminution (*see* p 200)

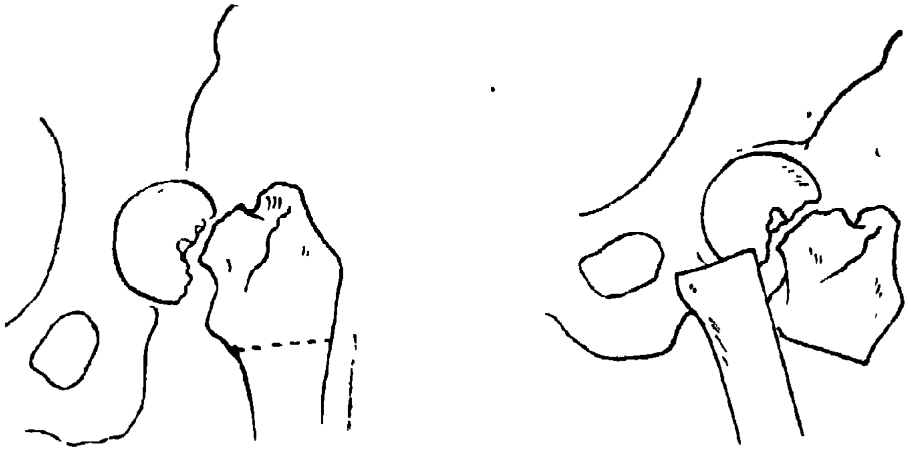

Fig 71.—Lorenz osteotomy.

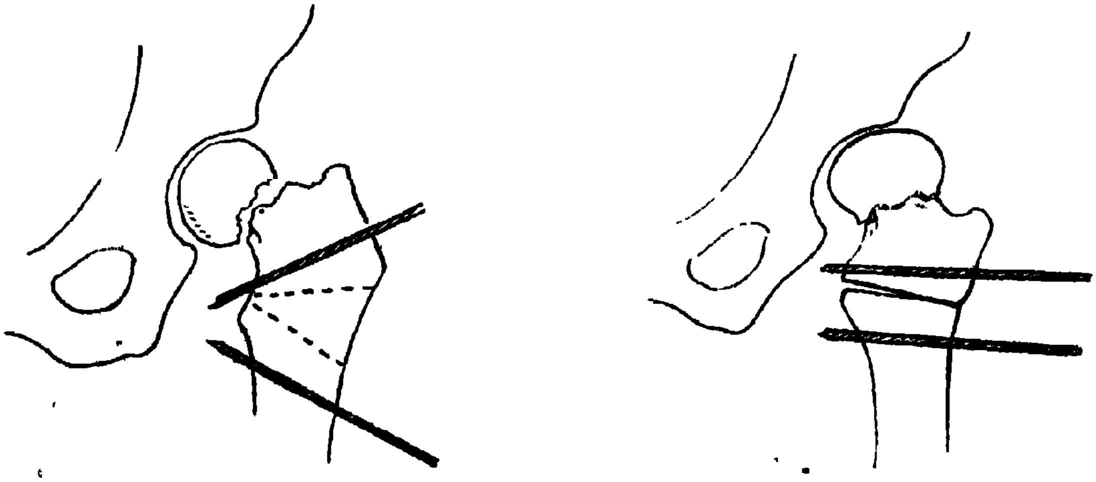

Fig. 72.—Schanz osteotomy.

Separation of the Upper Epiphysis.—The head does not join the shaft until eighteen. Its separation results from falls on the leg or hip. Occurs in patients of about fourteen. Its signs are very vague at the time. It results in marked coxa vara from the resulting reconstruction of the head of the bone (*see* p. 167).

TREATMENT.—Fixation in abduction of hip, or operation by a peg, followed by a walking calliper splint for six months.

Shaft.—

CAUSES.—Direct or indirect violence.

POSITION.—Upper, middle, or lower third

DIRECTION.—Transverse, oblique, or spiral.

DISPLACEMENT.—The lower fragment is always drawn up by the thigh muscles, and everted by weight of the limb

IN FRACTURE OF THE UPPER THIRD, the upper piece is flexed by the psoas and abducted; the lower piece is adducted.

IN FRACTURE OF THE MIDDLE THIRD, there is a tendency to backward sagging

IN FRACTURE OF THE LOWER THIRD, the lower fragment is flexed at the knee by the gastrocnemius, the upper fragment is driven down so as to penetrate the knee-joint or the quadriceps muscle and skin

Lower End.—

TRANSVERSE FRACTURE.—Similar to that of the lower third of the shaft Special liability to backward tilting of the lower fragment by the gastrocnemius This may cause an injury of the popliteal vessels.

T-SHAPED INTO THE JOINT

SEPARATION OF ONE CONDYLE.—Rare fractures. Crepitus on pressing one or both condyles. Marked effusion into joint

SEPARATION OF THE LOWER EPIPHYSIS.—Caused by hyperextension of knee in patients under twenty Epiphysis is displaced forwards The lower end of the shaft presses on the popliteal vessels

Treatment of Fractured Shaft of Femur.—

ESSENTIAL PRINCIPLES OF TREATMENT.—

1. The leg must be restored to full length, and this usually involves a stage of over-extension as a preliminary
2. Correct alinement must be attained.
3. Rotation outwards or inwards of the lower fragment must be prevented
4. The mobility of the knee must be preserved

THOMAS'S METHOD.—Extension straps are fixed (plaster or glue) to each side of the leg and thigh, and bandaged in place (*Figs*. 74, 75).

Leg is put up in a Thomas knee splint and traction made upon it until full length is attained.

The straps are tied to the end of the splint.

Kept in place with occasional tightening of straps for six or eight weeks, then replaced by a Thomas walking calliper splint (*Fig*. 73).

MODIFIED THOMAS'S METHOD.—

Extension strapping and splint as above.

Splint is bent at the knee to 135°, or a hinged piece is added (*Fig*. 76).

Leg in splint is slung to an overhead beam, or pulled on by a 15-lb weight tied to a cord running over a pulley (*Fig*. 77).

Foot is kept dorsiflexed by gauze glued to sole and tied to a wire frame fixed to splint.

Femur—Shaft—Treatment, ***continued.***

TRACTION AND SUSPENSION.—Adhesive plaster is fixed to the two sides of the leg with a wooden spreader below the foot to give attachment to a cord and weight. The leg is slung in semiflexion to an overhead beam (*Fig.* 78) Counter-traction is by a perineal band round the opposite leg. Is only suitable for young patients or those with poor muscular development

SKELETAL TRACTION —This should be used for all adult cases with much displacement

1 Transfixion by a taut wire (Kirschner) held in a steel horse-shoe (*Fig* 79)

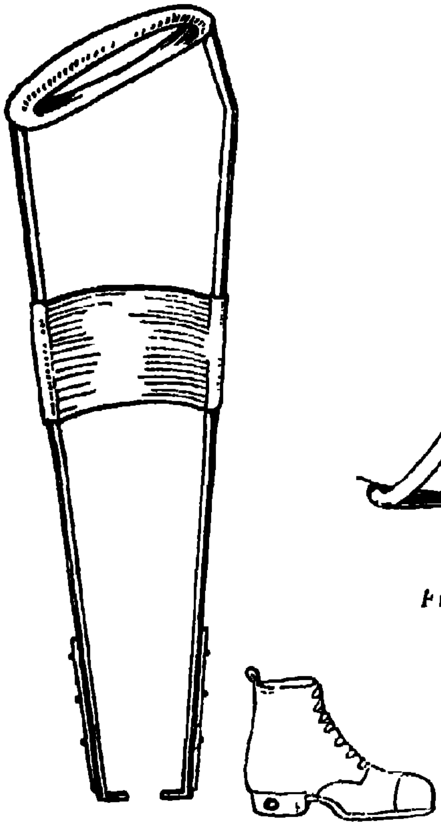

Fig. 73. Walking calliper splint slotted into heel of the boot.

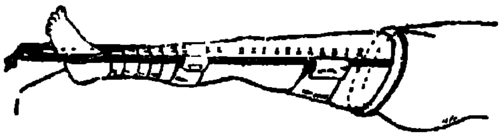

Fig 74.—Fixed Thomas's splint.

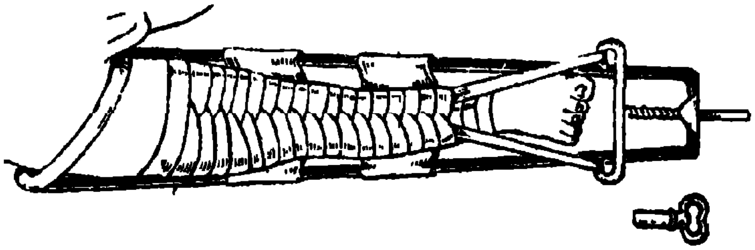

Fig 75.—Fractured femur treated by Thomas's splint, provided with screw traction

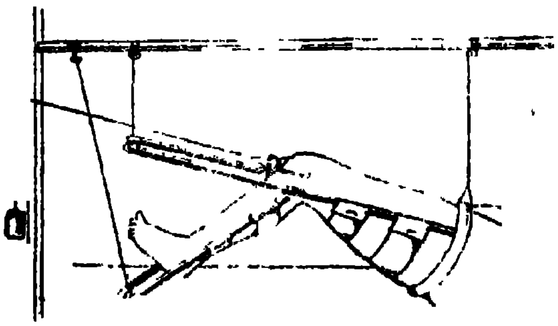

Fig 76.—Thomas's splint with hinged attachment at knee-joint.

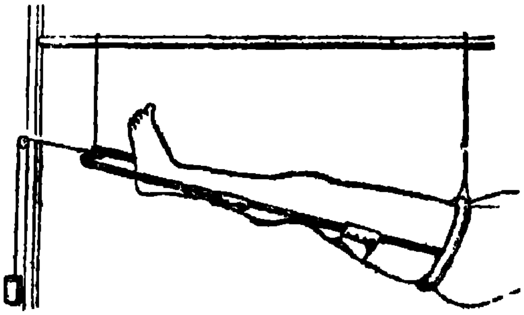

Fig 77.—Slung Thomas's splint.

The wire is inserted by some surgeons through the lower end of the femoral shaft, especially in fracture of the upper third of the femur (*Fig.* 80). This may lead to stiffness of the knee, following the formation of adhesions round the joint due to low-grade sepsis along the track of the wire.

In fractures of the upper and lower third, the transfixion should be through the crest of the tibia just below the tubercle. In this position a steel pin is quite as good as the wire and simpler in its application. (*Fig.* 81.)

2 The Weight used should be enough to produce over-distraction of the fragments; 25 lb is the most usual It is better to put on a big

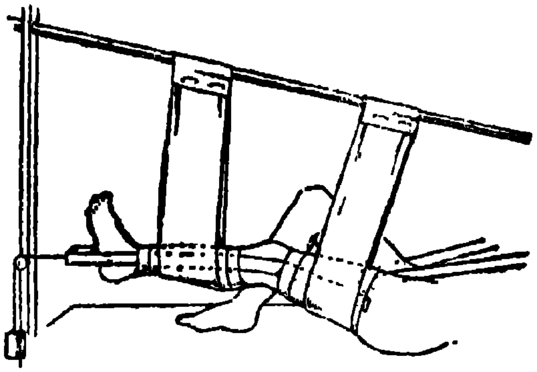

Fig 78.—Balkan beam. Traction by adhesive plaster

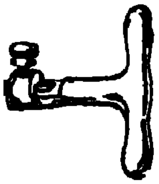

Fig. 81 — Transfixion pins and handle (×½)

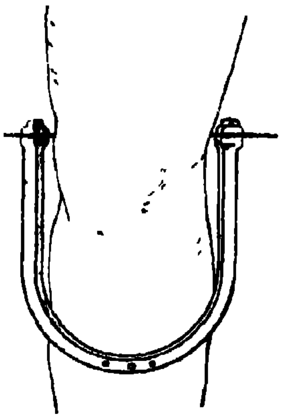

Fig. 79.—Kirschner's taut wire method

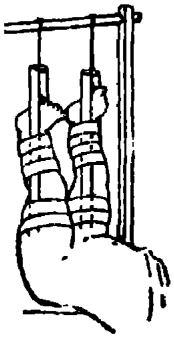

Fig 82.—Child's legs slung to a gallows splint.

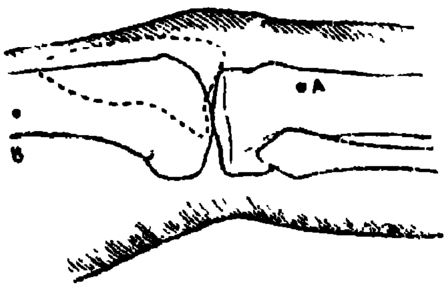

Fig. 80.—Points of transfixion of femur (B) and tibia (A). Dotted lines show limit of synovial membrane.

Femur—Shaft—Treatment, ***continued.***

weight and then to reduce after a few days, than to begin with a small weight and then to increase. As much as 40 lb. may be used in an old case (i.e., where treatment has been delayed). A weight of 15 lb. on the femur or 20 lb. on the tibia can be tolerated for six weeks. If the transfixion site becomes inflamed, adhesive plaster can be substituted for the skeletal traction after the latter has got the femur into good position.

3. POSITION OF THE LEG.—The leg should always be slung to an overhead beam or rested on a cradle splint in a position of abduction of the hip and flexion of the knee (*Fig.* 83). Abduction is most important in fractures of the upper third, flexion of the knee in fractures of the lower third
4. ACTIVE MOVEMENTS.—Flexion and extension of hip by patient lifting himself up and lying down in bed Movements of knee, by supporting the knee by a rest, and the patient flexing and extending by help of a cord and pulley from the foot to an overhead pulley. Faradic stimulation of the quadriceps is of value to maintain quadriceps tone. Patella must be kept mobile on the condyles.

FRACTURES AT THE LOWER END, INVOLVING THE CONDYLES. —Accurate reduction of the articular end is essential. Usually done best by skeletal traction through tibial tubercle, the knee being flexed to a right angle. If this fails, an open operation may be needed, the two condyles being plated together (*Fig* 86).

AFTER-TREATMENT.—Bony union takes about six weeks, but consolidation will need about three to six months. The progress of union of the fracture and the position must be ascertained by frequent X-ray control, e.g., every two weeks. Traction is discontinued after six weeks. When the patient gets up the leg should be supported by a Thomas's walking calliper. This is better than any form of fixed splint or plaster case, because it can be removed daily for exercises

IN CHILDREN —Both legs are slung to an overhead bar so as to raise the buttocks off the bed (*Fig.* 82).

OPEN OPERATION.—For following cases.—

Where efficient pin or calliper traction has failed to reduce, due to interposition of the soft parts

Certain spiral fractures and those where a third fragment lies crosswise and cannot be reduced by traction

For T-shaped fractures into knee-joint with marked displacement.

ADVANTAGE OF OPEN OPERATION —

Accuracy of reduction and fixation

DISADVANTAGES OF OPERATION.—

Risks of an open operation

Return to full function is much slower than after equally efficient closed traction methods.

Plates and screws often cause late irritation and have to be removed.

CHOICE OF OPERATION.—

Long oblique or spiral fractures: Fixed by vitallium screws (*Fig.* 84).

Transverse fracture without comminution Intramedullary bone peg.

Other cases: Plates and screws or bolts (*Figs.* 85, 86).

Prognosis after Fracture of the Femur.—

GOOD.—In young patients. If reduction has been perfect. If mobility of the knee-joint has been preserved. Full recovery in six months.

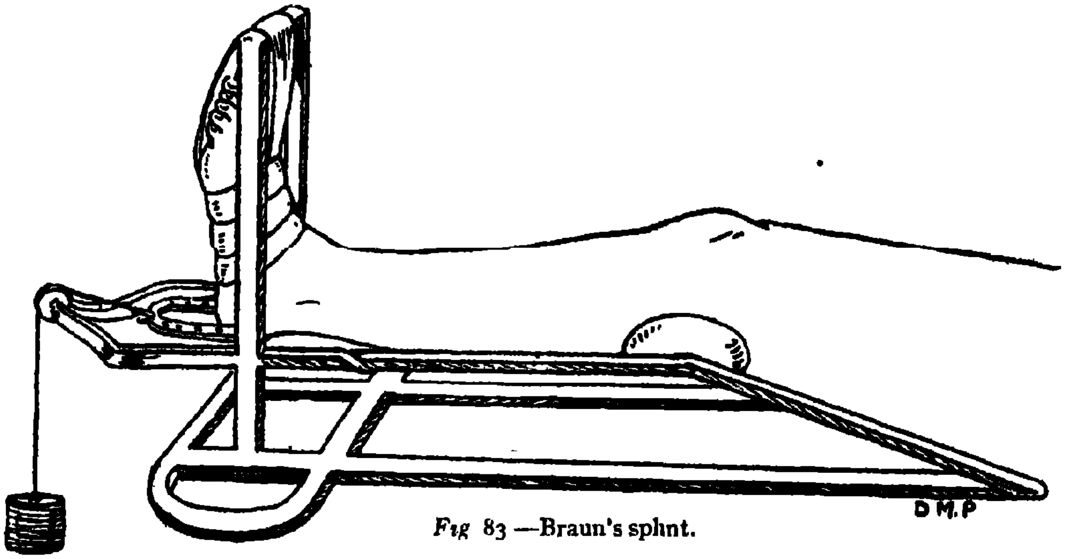

Fig 83 —Braun's splint.

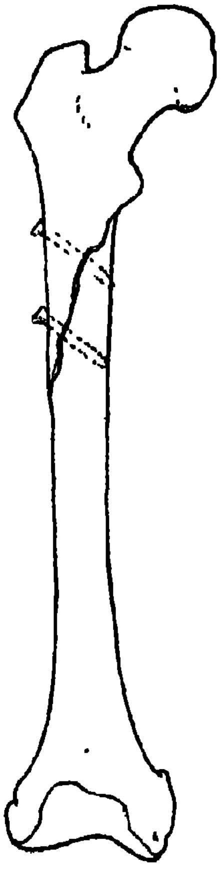

Fig. 84. — Oblique fracture treated by two vitallium screws

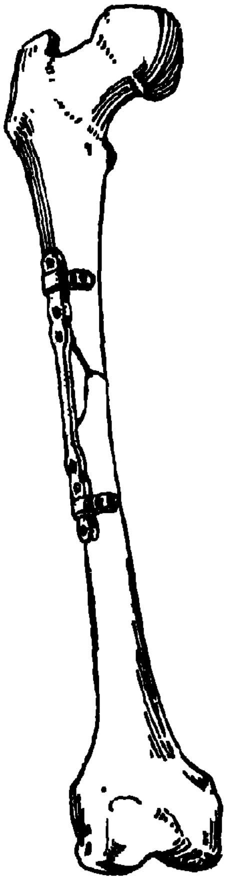

Fig. 85. — Butterfly fracture of shaft treated by clipped plate.

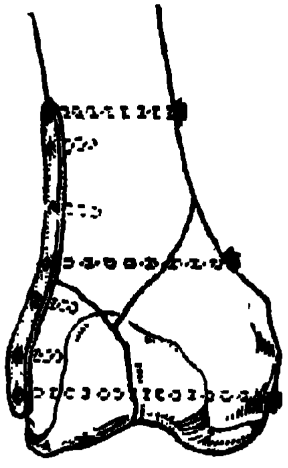

Fig. 86.—T-shaped fracture of condyles treated by bolted plate.

Femur—Prognosis after Fracture, *continued.*

DOUBTFUL, with slow recovery in twelve months or more If knee has become stiff, or if by neglect of active exercises during treatment the muscles have wasted.

BAD, if mal-union, shortening, angulation, or rotatory deformity persist. Return to heavy work is impossible.

PATELLA

CAUSES.—

DIRECT VIOLENCE —Usually produces a stellate or fissured fracture The aponeurosis is not separated. Little or no displacement results.

INDIRECT, i.e., muscular violence of the quadriceps extensor This is the common form to which the following applies. The patella is broken across the femoral condyles by force applied to its two ends and the quadriceps tendon is torn across

ANATOMY.—Marked displacement of the fragments in the transverse fractures. The ragged aponeurosis of the quadriceps hangs over the divided bony ends. Hæmorrhage and effusion into the joint If quadriceps expansion not torn then no displacement occurs

SIGNS.—Inability to extend the knee. Presence of the upper fragment high above the knee. Gap between the fragments.

UNION.—By fibrous tissue forming a band between the fragments Absence of bony union is due to. (*a*) Interposition of aponeurosis between the fragments; (*b*) Muscular traction separating and tilting the fragments.

TREATMENT.—Essential step consists in accurate suture of the torn aponeurosis at each side of and over the patella In cases without separation immobilize with knee in extension in a plaster-of-Paris splint from groin to toes

REMOVAL OF THE BROKEN FRAGMENTS.—This makes suture much easier and more accurate Only a firm bandage over a woollen covering is necessary afterwards The patient should be up and walking by the end of one week and back at work at the end of one month

Note.—The quadriceps tendon only passes over the patella and is not inserted into it. The joint is actually stronger and more mobile when the patella has been removed

WIRING THE PATELLA.—This has been the classical method since Lister's pioneer work. But it gives a slow recovery and often leads to limitation of movement, owing to incomplete apposition, or to breaking of the wire. As an alternative, catgut can be used, or a fascial strip can be turned down from the iliotibial band to encircle and approximate the fragments.

TIBIA AND FIBULA

Tibia.—

AT UPPER END.—Generally results from direct violence Sometimes T-shaped into the joint. Not much displacement. One tuberosity only may be broken off from the shaft, whilst the opposite collateral ligament is torn. Marked genu valgum or varum will result. Fracture of the external tuberosity may be of two types: (1) Depressed fracture without comminution; (2) Comminuted fracture with separation of the tuberosity.

TREATMENT.—Fixation until union has occurred in a plaster cast or Thomas's splint in position of full extension of knee. Followed by a calliper splint hinged at the knee; this is removed daily for active exercise of the knee-joint. If the lateral ligament has been torn, it is wise to continue this for two or three months. Accurate reduction is essential; depressed fractures have to be elevated and maintained reduced by some means of internal fixation. Böhler's redresseur may be necessary to bring about reduction. Essential to institute early quadriceps drill.

OF THE TUBERCLE.—This may ossify from a separate nucleus, which appears about twelve and joins shaft at fourteen. Generally grows down as a beak from the head. Generally detached by muscular action of the quadriceps. Sometimes by direct violence.

TREATMENT.—By plaster in position of extension, or nail if there is much separation.

OF THE SHAFT.—Caused by direct blows Transverse above and oblique below. Irregularity of anterior border. Pointed end of upper fragment may pierce the skin.

TREATMENT.—By plaster cast with walking iron (*Figs*. 87, 92)

OF THE INTERNAL MALLEOLUS.—From a blow on the ankle, or from a wrench of the foot inwards. Likely to be followed by a stiff ankle.

TREATMENT —As for fracture-dislocation at the ankle-joint

Fibula.—

SIGNS are: (1) Localized pain on pressing the two bones together; (2) Loss of the fibula spring

TREATMENT.—By plaster cast and walking iron (*Figs* 87, 92)

Tibia and Fibula.—

WHEN FROM DIRECT VIOLENCE —Bones are broken at the same level. Fracture is transverse. Not much displacement Great contusion.

WHEN FROM INDIRECT VIOLENCE.—Tibia breaks at junction of middle and lower third. Fibula breaks higher up. Fracture is oblique downwards, forwards, and inwards Great displacement Lower fragment drawn up and rotated outwards Upper fragment often pierces the skin

Spiral-shaped fracture is produced by torsion Is specially difficult to reduce

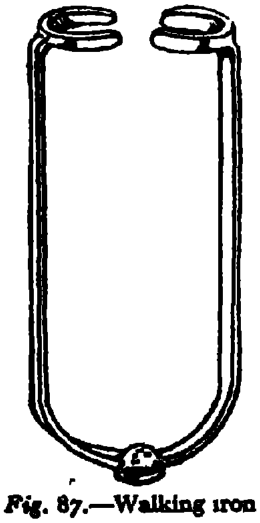

Fig. 87.—Walking iron

Tibia and Fibula, ***continued***

TREATMENT.—

In cases with much displacement, skeletal traction from a pin passed through the os calcis is the method of choice. The leg is slung on a cradle or Braun's splint (*Fig*. 83). If the fracture of the tibia is oblique, after reduction has been effected, a second pin is passed through the crest of the tibia, below the tubercle, the whole leg is encased in a plaster cast with a walking iron (*Fig*. 87), and the patient allowed up. Plaster kept on for six to eight weeks.

Open operation is indicated in young muscular subjects whose skeletal traction has failed to attain good reduction This often indicates the interposition of soft parts. Reduction is effected through a short anterior incision. A transfixion pin is passed through both fragments, and the projecting ends of this are incorporated in the walking plaster, The pin can be removed after two to three weeks, whilst the plaster is left on for six to eight weeks. Alternatively, bone ends can be maintained in apposition by a stainless steel screw

Fractures at the Ankle-joint.—

CAUSED BY indirect violence—Tripping or turning the foot

DISPLACEMENT —Foot displaced and rotated

VARIETIES.—Tendency to classify all fractures in the region of the ankle-joint as Pott's fractures—this is not correct The abduction fracture at the ankle-joint is the type that was described by Pott Classified as follows —

1. EXTERNAL ROTATION INJURIES —Torsion causes a spiral fracture of the external malleolus Three degrees · (*a*) Fracture of fibula—oblique from the tibio-fibular joint (*Fig*. 88) (*b*) As (*a*), above, but with avulsion of the internal ligament or fracture of internal malleolus Outward dislocation of joint (*c*) As (*b*), but with posterior marginal fracture of tibia
2. ABDUCTION FRACTURES — (*a*) Fracture of internal malleolus or avulsion of internal lateral ligament (*b*) As (*a*), but with fracture of fibula (transverse) (*Fig*. 89). (*c*) As (*b*), but with fracture (marginal) of lower end of tibia, or rupture of tibio-fibular ligament—i.e , tibio-fibular diastasis (*Fig*. 90)
3. ADDUCTION FRACTURES —(*a*) Fracture of internal malleolus (vertical). (*b*) As (*a*), but with fracture of external malleolus. (*c*) As (*b*), but with posterior marginal fracture and backward dislocation of the joint (*Fig*. 91)
4. VERTICAL COMPRESSION FRACTURES.—Due to a fall from a height, and there is an anterior marginal fracture of the lower end of tibia.

Abduction and external rotation account for the majority of fractures at the ankle-joint

SIGNS.—Great swelling from bleeding and effusion into the ankle-joint.

Foot is displaced, generally outwards and slightly backwards, and rotated outwards.

Internal malleolus is very prominent. Fracture of the fibula three or four inches above ankle.

Crepitus may be felt, and there is loss of the fibula spring.

TREATMENT.—

REDUCE UNDER ANÆSTHETIC.—Traction with rotation of the foot, pulling the foot forwards. Keep the knee bent to relax the gastrocnemius.

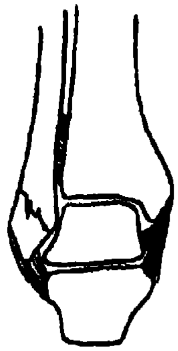

Fig. 88.—Common form of ankle fracture

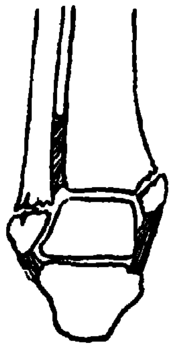

Fig. 89.—Pott's fracture—first degree dislocation.

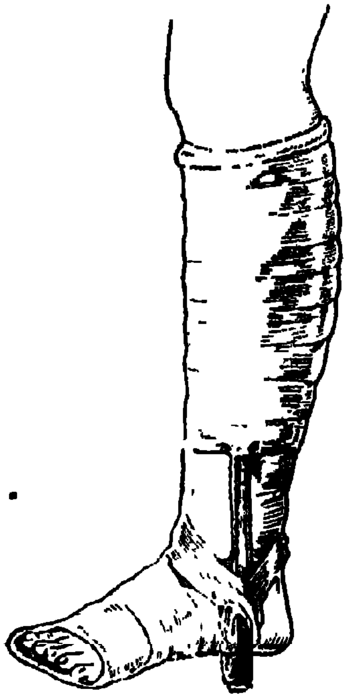

Fig. 92.—Leg treated with plaster cast and walking iron for below-knee fractures.

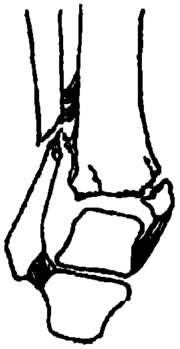

Fig. 90.—Abduction fracture. Third degree dislocation

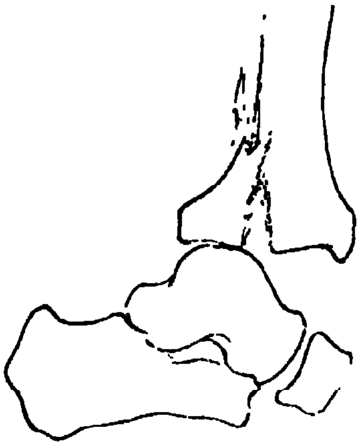

Fig 91 —Adduction fracture Third degree dislocation.

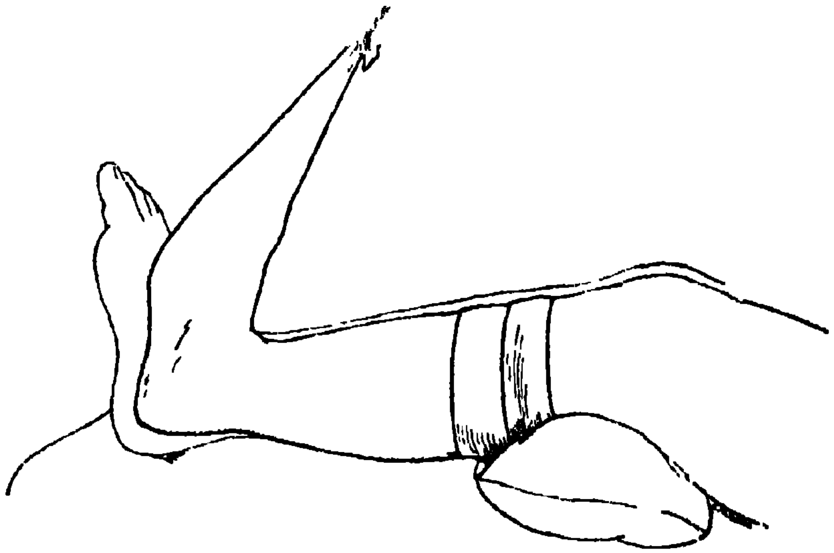

Fig. 93.—Stocking suspension for fracture about the ankle-joint. The knee is slightly flexed by slinging or by a pillow. A piece of stockingette is placed on the leg and its upper edge fixed to the skin by adhesive plaster. The fracture-dislocation having been fully corrected, the foot is slung to an overhead support so as to secure (*a*) dorsiflexion, (*b*) forward position of the foot, and (*c*) inversion.

Fractures at the Ankle-joint—Treatment, *continued*

Put up in a plaster case from the tubercle of the tibia to the toes, leaving the dorsal surface of the toes free Incorporate a walking iron (*Fig.* 92).

In Cases with Much Swelling — After reduction, elevate on a cradle or Braun's splint (*Fig* 83), or simply use a stocking suspension (*Fig.* 93) until swelling has subsided and then put up in plaster.

In Cases which are sent Home after Setting.—The plaster case should be split down the front, and the patient instructed to return at once if pain or swelling of the toes occurs

In Cases of Tibio-fibular Diastasis the inferior tibio-fibular joint should be immobilized by insertion of a vitallium screw through tibia and fibula at lower end

Cases with Marked Dislocation of the Ankle and fracture of the posterior or anterior tibial margin —Preliminary skeletal traction through the os calcis. When reduction is complete, a second pin through the crest of the tibia, plaster cast and walking iron (*Fig.* 92)

In Cases with Mal-reduced Fractures, arthrodesis of the ankle-joint may be necessary

Vertical Compression Fractures need accurate reduction of the fragments and maintaining immobilization by a vitallium screw

After-treatment.—On removal of the plaster cast after four, to six weeks a covering of Unna's paste or an Elastoplast bandage should be used for some weeks Alternatively provide patient with a valgus iron.

In cases with the original outward displacement of the foot, it is a wise precaution to use a shoe with a raised inner edge of the sole and heel, so as to relieve the strain on the ligaments on the inner side of the foot and prevent valgus deformity

Prognosis after Lower Leg Fractures.—

GOOD, with ability for hard work within three months.—

Fracture of one bone only, with little displacement

Fractures accurately reduced, firmly fixed, and exercised

DOUBTFUL.—Return to work in twelve months or more.

If ankle has become stiff in dropped position; if exercises have been neglected.

BAD.—Never return to laborious work

Displacement unreduced, especially in or near the ankle-joint

Any angular deformity of the shafts, throwing ankle out of line.

OS CALCIS

Caused by falls from a height on to the feet

Various degrees of comminution Typically the waist of the bone is driven downwards and the sides splayed outwards (*Fig.* 94). May get fracture of body of bone of the compression type, fracture of the posterior portion—traction fracture due to muscular violence—or fracture of the medial tuberosity. In compression fractures involving the subastragaloid joint there is an alteration in the salient angle of Böhler—normal angle 30°.

Treatment.—Screw traction through a pin in posterior process of the os calcis.

Pressure of the sides of the bone by a screw clamp.

Fixation in a plaster cast with double transfixion pins (*Fig.* 95).

Prognosis is bad if the displacement has not been reduced May be necessary to arthrodese the subastragaloid joint.

ASTRAGALUS

A rare fracture. Seen more commonly now when aircraft crash—"rudder bar" fracture. Fractures of the neck may occur, with or without subtaloid dislocation, and posterior dislocation of the body. Avascular necrosis may follow the latter injury. Bone usually broken into an anterior and posterior portion and complicated by dislocation of nearby joints.

TREATMENT.—Reduce if possible—walking plaster as in Pott's.

If reduction is impossible—open operation, fix or remove displaced fragment.

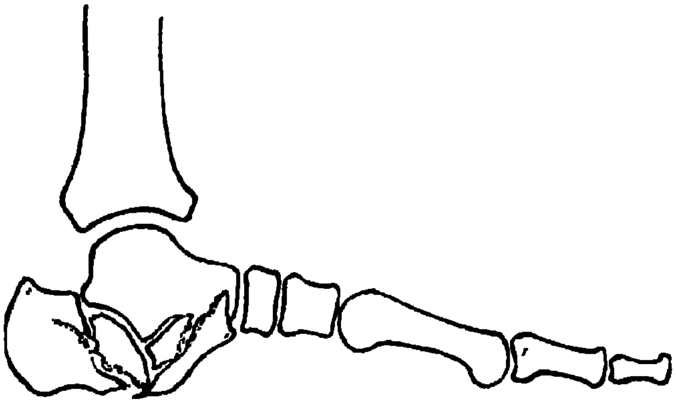

Fig. 94.—Crush fracture of the os calcis

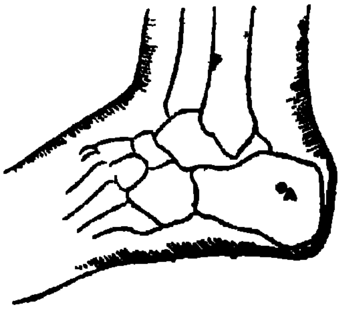

Fig. 95.—Point of transfixion of os calcis.

CHAPTER XXI

DISEASES OF BONES

INFLAMMATORY DISEASES OF BONES

Structure of Bones.—

HARD PARTS.—

DENSE BONE forms shaft of long bones, outer and inner tables of flat bones, and outer shell of short bones.

CANCELLOUS BONE forms interior of epiphyses and epiphysial ends of shafts of long bones, also interior of short and flat bones.

SOFT PARTS.—

PERIOSTEUM.—A vascular membrane outside bones, with deep layer of growing cells It ceases where the epiphysis joins the shaft.

MEDULLA.—A very vascular and lymphatic mass of cellular tissue inside the cancellous tissue and in shaft of long bones

HAVERSIAN CANAL SYSTEM —Minute vessels which traverse the dense bone, and unite periosteal with medullary vessels

CARTILAGE —

Epiphysial—A vascular layer of growing cartilage between epiphysis and diaphysis, from which the growth of the shaft in length takes place. Its circumference is connected with the periosteum.

Articular.—Smooth cartilage covering articular ends of the diaphysis. Non-vascular.

The metaphysis is the growing end of the bone just on the shaft side of the epiphysial cartilage.

VESSELS.—

NUTRIENT ARTERY, with vein and lymphatics, pierce shaft and end in the medulla

PERIOSTEAL VESSELS supply the periosteum and send inward branches to Haversian canals.

EPIPHYSIAL VESSELS, given off from the vascular anastomosis round the joint to the epiphysis, especially at the junction with the shaft, where they supply the epiphysial cartilage.

HAVERSIAN VESSELS —Minute twigs which join the periosteal with the medullary vascular system.

LYMPHATICS are present in the perivascular sheaths.

Relation of Inflammatory Processes to Bone Tissues.—

ORIGIN —Inflammation always begins in the vascular soft tissues, i.e., in periosteum, medulla, or metaphysis.

EXTENSION.—Inflammatory processes spread from periosteum to medulla, or from medulla to periosteum, by: (1) Haversian canal system; (2) Vessels of the metaphysis.

THE HARD PARTS of the bone may be regarded as intercellular material, which reacts in three different ways to inflammation:—

1. NECROSIS.—Death *en masse* from acute inflammation with vascular thrombosis.

2. CARIES.—Molecular necrosis or ulceration, from subacute inflammation.
3. SCLEROSIS.—From chronic inflammation laying down new bone.

Necrosis is caused by:—

Periostitis: Acute localized—Acute infective—Chronic syphilitic

Osteomyelitis: Acute localized or septic—Acute infective—Subacute septic, tuberculous, syphilitic.

Toxic poisoning: Phosphorus—Mercury.

Senile necrosis, corresponding to senile gangrene

Avascular necrosis is a traumatic form, resulting from cutting off of the blood-supply to a portion of the bone, e.g., the head of the femur in subcapital fractures.

Necrosis corresponds to gangrene or sloughing of soft parts.

Caries.—An inflammatory rarefaction of bone: Simple (as in repair of fractures)—Septic (inflammation of cancellous tissue)—Tuberculous (the commonest form of caries)—Syphilitic.

CARIES SICCA.—Caries without suppuration

CARIES SUPPURATIVA.—Caries with suppuration

CARIES FUNGOSA.—Caries with exuberant granulations

CARIES NECROTICA—Caries associated with necrosis of minute portions of bone: corresponding to ulceration with sloughing of soft parts.

Caries corresponds to ulceration of soft parts, and fibroblasts of granulation tissue are represented by multinuclear cells called osteoclasts, which eat into and absorb the cancellous bone

Sclerosis results from chronic inflammatory processes: Chronic periostitis—Chronic osteomyelitis.

SIMPLE—in the proximity of some inflammatory focus in soft parts, e.g., chronic ulcer over tibia

SYPHILITIC—much the commonest

TUBERCULOUS—rare; beyond area of tuberculous affection

Sclerosis corresponds to fibrosis of soft tissue, and results from a laying down of fresh bone (corresponding to intercellular fibrous tissue) by medulla, periosteum, and Haversian vessels

PERIOSTITIS

VARIETIES—

ACUTE LOCALIZED

ACUTE DIFFUSE OR INFECTIVE.—Generally associated with infective osteomyelitis as cause or effect

CHRONIC.—Simple—Tuberculous—Syphilitic (local, general)

Acute Local Periostitis.—

CAUSES—Traumatism—Specific fevers—Pyæmia—Alveolar abscess—Gout

RESULTS—(1) Resolution, (2) Chronic thickening, (3) Suppuration with necrosis

PATHOLOGY.—

IN ASEPTIC CASES—Exudation from deep layer of periosteum. Organization of this exudation.

IN SEPTIC CASES—Exudation from deep layer of periosteum. Thrombosis of vessels. Suppuration of the exudation. Rupture of vessels passing from periosteum to underlying bone, caused by tension of exudation Necrosis of fragment of bone which is separated from its periosteum.

Acute Local Periostitis—Pathology, *continued.*

Necrosed fragment is called the SEQUESTRUM.

Living bone round sequestrum is rarefied by the inflammatory proliferation of cells forming GRANULATION TISSUE.

REACTION BETWEEN GRANULATION TISSUE AND SEQUESTRUM may be:—

1. Absorption of sequestrum if small and sepsis is at an end.
2. Erosion of sequestrum by granulations until it is separated from living bone.
3. If sepsis is still active, the living bone will be absorbed by granulation tissue until sequestrum lies free. Occupies about four to eight weeks

Deep surface of separated periosteum in the meantime forms new bone—the INVOLUCRUM

Pus under the periosteum escapes through periosteum and soft tissues. This opening remains in the involucrum, and is called a CLOACA

The sequestrum escapes through the cloaca when it is loose, and if the cloaca is large enough.

After the escape of the sequestrum, the granulation tissue lining the living bone becomes ossified, forming a dense layer of osteosclerosis.

TREATMENT.—As for osteomyelitis.

Acute Diffuse or Infective Periostitis is practically the same disease as ACUTE INFECTIVE OSTEOMYELITIS (*see below*), the one being the cause or effect of the other

Chronic Periostitis.—*See* CHRONIC OSTEOPERIOSTITIS (p 219)

OSTEOMYELITIS

VARIETIES —

ACUTE INFECTIVE —Associated as cause or effect with infective periostitis.

TRAUMATIC —Resulting from infection of an open fracture

SUBACUTE —Simple or infective—Associated with repair of fractures, and with separation of sequestra

CHRONIC —Simple—Tuberculous—Syphilitic.

EPIPHYSITIS.—An acute infective osteomyelitis of the epiphysis

Acute Infective Osteomyelitis (*Figs* 96, 97, 98) —

AETIOLOGY and PATHOLOGY.—

CHILDREN, five to fifteen being the commonest age, that is, when the epiphysial cartilages are in full activity.

Supervenes on a condition of general DEBILITY.

Often follows EXANTHEMATA, e g , scarlet fever.

Often follows SEPTIC ULCERS of mouth, throat, or intestines

Generally results from a BLOW or WRENCH.

Caused by entrance of PYOGENIC MICRO-ORGANISMS.—

Staphylococcus pyogenes aureus. Common type.

Staphylococcus pyogenes albus Mild type.

Streptococcus pyogenes Young children. Very fatal Less extensive necrosis.

Pneumococcus.

Bacillus coli communis, in mixed infection

PRIMARY BONY FOCUS is usually the growing vascular tissue on the shaft side of epiphysial cartilage, called the metaphysis

VARIETIES of infective osteomyelitis:—

1 **Medullary Osteomyelitis.**—Medulla becomes inflamed, the veins thrombosed, and pyæmia often follows. Haversian vessels are infected. Bony walls of Haversian canals being unyielding, the exudation causes strangulation of vessels. This cessation of circulation and the toxins kill the bone, producing total necrosis. Periosteum is infected Its deep layers suppurate, and it is stripped off the shaft. Epiphyses escape because.—

a. They have an independent blood-supply
b. They are separated from the shaft by a comparatively avascular zone of cartilage
c They have no periosteum through which infection can take place

2. **Acute Arthritis** —Suppuration will extend into the neighbouring joint —

a If the epiphysial cartilage lies within the joint capsule, e g., hip or elbow
b. In infants, where epiphysis consists of thin cartilage only, this may be perforated, pus so reaching joint cavity
c. By extension along soft tissues, e g , biceps tendon into the shoulder-joint

3. **Chronic Abscess** —Where infection is mild and resistance is good, the inflammatory process may be limited, and an abscess forms in the cancellous tissue, surrounded by dense thickened layer of osteosclerosis. Commonest at upper end of tibia, lower end of femur, or lower end of tibia (*Fig* 99) A similar abscess may result from tubercle (often termed 'Brodie's' abscess, after the surgeon who first described it). A large majority are of staphylococcal origin

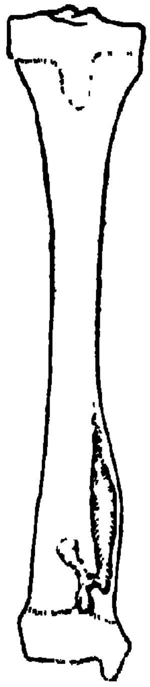

Fig. 96.

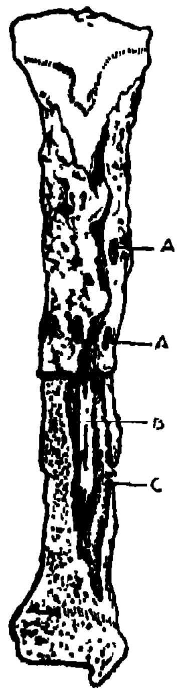

Fig. 97.

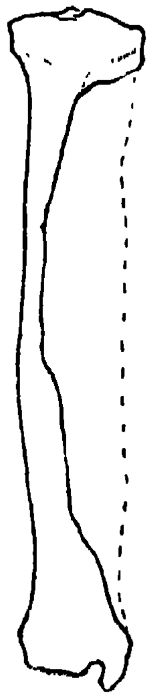

Fig 98

Fig 96 - Osteomyelitis, early stage Infective focus at junction of diaphysis and epiphysis. Pus has spread to the surface of the bone and has partly stripped the periosteum.

Fig 97 —Osteomyelitis, late stage A massive sheath of new inflammatory bone (the involucrum) has formed round the original shaft, which now lies dead as a sequestrum (B). A cavity filled with pus (C) lies between the sequestrum and the involucrum, and communicates with the exterior by apertures (A) in the latter, called cloacæ.

Fig. 98. —Osteomyelitis. Diagram showing method of treatment in late stages. The involucrum is cut away on one side of the bone, and the sequestrum removed.

Acute Infective Osteomyelitis, *continued.*

TERMINATION.—Apart from complications, all varieties except the last two present three stages:—

1. STAGE OF INFECTION and suppuration, when the periosteum is stripped off all or a part of the shaft.
2. STAGE OF PERIOSTEAL OSSIFICATION.—The pus escapes through one or more openings in the periosteum and soft tissues. These are the cloacæ. The deep layer of the periosteum produces a shaft of new bone—the involucrum.
3. STAGE OF SEPARATION OF THE SEQUESTRUM.—Sequestrum is separated from the living bone (in total necrosis this will be at its two ends). This occupies about four weeks in the case of small bones, up to three months in the femur

SYMPTOMS —Rigor, with high fever Severe pains in the limb. Brawny swelling of soft parts. Absence of the scarlet flush of superficial inflammation Tenderness is very great and limited to one spot over the bone. Joint can often be moved gently without pain. Severe toxic symptoms.

COMPLICATIONS —

LOCAL —Acute arthritis (*see* p. 217). Sloughing of the periosteum, preventing the regeneration of the bone. Destruction of epiphysial cartilage, stunting growth Overgrowth of the bone by stimulation of chronic inflammation and long-standing hyperæmia.

GENERAL —Septicæmia—Pyæmia—Amyloid disease due to prolonged suppuration—Spontaneous fracture of the bone

PROGNOSIS —Always grave It is severe in proportion to Youth of patient—Debility of patient—Size of the bone—Extent of the bone disease—Virulence of infection.

Streptococcal infection is almost hopeless. *Staphylococcus aureus* infection is grave *Staphylococcus albus* infection is milder

TREATMENT —

GENERAL TREATMENT suitable for fever. Chemotherapy with sulphathiazole when the condition is due to a staphylococcus has produced conflicting results Penberthy and Wheeler concluded that with correct chemotherapy in conjunction with early and adequate surgical drainage of the lesion, the mortality from septicæmia can be greatly reduced, and, secondly, chemotherapy limits the amount of bone destruction and the appearance of secondary metastatic lesions. Again, chemotherapy alone, with immobilization of the limb in a

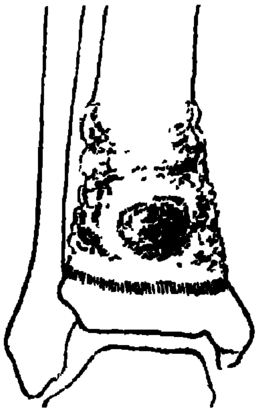

Fig. 99 —'Brodie's abscess' of the tibia. A chronic staphylococcal infection at the lower end of the bone arising at the epiphysial junction and spreading out towards the periosteum. If not treated early it causes massive thickening of the bone.

plaster shell, has cured certain cases of osteomyelitis, X-ray control showing revascularization of the necrosed bone. Unfortunately some staphylococci are not affected by chemotherapy, so surgical treatment cannot be excluded completely. Penicillin has proved valuable.

OPERATION —Lately doubt has been thrown on the wisdom of early operation. Some would even advise no operation unless an abscess points. Probably some delay in operating will give better results.

SIMPLEST OPERATION.—In early acute cases: incise periosteum, drill several holes into metaphysis.

CLOSED VASELINE PACK.—The cavity in the bone is made as wide and shallow as possible (like a saucer), and the soft parts are drawn aside The whole cavity after swabbing with iodine and spirit is packed with vaseline gauze without any sutures. The whole limb is encased in plaster-of-Paris so as to give absolute fixation of the joints above and below the inflamed bone. No window is cut. The plaster is left for 4 to 8 weeks in spite of unpleasant smell. Only increase of pain or persistent rise of temperature requires the plaster to be cut. The wound toilet, vaseline pack, and plaster are renewed at intervals of 6 to 8 weeks, and after about two or three such renewals healing will have occurred. This treatment saves many months in hospital, and many painful dressings. (Winnett Orr)

DIAPHYSECTOMY.—Total removal of the affected diaphysis is never justified except for the clavicle and fibula If done for other long bones it will lead to non-regeneration

It total necrosis has occurred, cut sequestrum in two and twist each end off from epiphysis· except in case of humerus, femur, or tibia, when sequestrotomy must be delayed until the involucrum has formed.

When involucrum has formed, wait until examination by the probe shows that the sequestrum is loose, then enlarge cloacæ and remove sequestrum.

AMPUTATION may be needed: When sequestrum is inaccessible, e g., at the back of lower end of the femur—When pyæmia is present—When exhaustion threatens life—When suppuration in a large joint occurs—When lardaceous disease has begun

Traumatic Osteomyelitis.—

CAUSES.—Septic open fractures—Amputations—Any bone operation.

SYMPTOMS.—Inflammation and suppuration of wound—End of the bone is seen or felt to be white and bare.

High temperature, with rigors or pyæmia, may occur

Portion of the bone is extruded as the sequestrum.

SEQUESTRUM: Tubular or conical It outer surface is pitted by granulations

TREATMENT.—Open up wound. Scrape out medullary tissue and drain. Amputate if pyæmia persists.

CHRONIC OSTEOPERIOSTITIS

Usually primarily a periostitis, but by extension the disease affects the Haversian canals and medulla, and produces a general sclerosis and thickening of the bone.

LOCAL.—Caused by local periostitis, e.g., beneath a chronic ulcer, or by trauma, rheumatism, syphilis. A hard node is formed by new periosteal bone, in which the Haversian canals run at right angles to the surface Later this becomes sclerosed.

Chronic Osteoperiostitis, *continued.*

DIFFUSE.—As an extension from the local variety

In syphilis as a primary diffuse periostitis or round a central gumma.

In tubercle from a central abscess or other chronic focus.

SYMPTOMS.—Deep aching pain, worse at night Bone is felt to be thickened, and seen to be so by the X rays.

TREATMENT.—Rest, counter-irritation, with iodides internally

If the pain is severe, an operation may be necessary. The bone is exposed, the periosteum stripped off, and the medulla opened by the trephine or gouge

Amputation may be required in the worst cases

TUBERCULOUS DISEASE OF BONES

Varieties of Tuberculous Disease.—

1 MILIARY TUBERCLE.—Very rarely found in the bones in general infection

2 CASEOUS FOCI.—Usually situated towards the end and near the surface The following zones occur from within outwards.—

a. A MASS OF CASEOUS MATERIAL occupies the centre. This represents the first seat of the disease, the cells of which have died and undergone fatty degeneration.

b. GRANULATION TISSUE containing tubercles in which all bony trabeculæ have been absorbed

c A ZONE OF SCLEROSIS, containing small round cells instead of fatty tissue

d A ZONE OF RAREFYING OSTEITIS at some distance from the deposit.

The whole process spreads to the periphery, whether joint or periosteal surface.

3 NECROSIS WITH SEQUESTRUM FORMATION.—This also occurs chiefly in the ends of long bones. The sequestra vary in size, and are often wedge-shaped, with the base of the wedge towards the joint Here three zones occur, viz. —

a. THE CENTRAL SEQUESTRUM, in which the trabeculæ are thickened

b A ZONE OF TUBERCULOUS GRANULATIONS.

c. A ZONE OF RAREFYING OSTEITIS

The formation of caseous foci and of sequestra often occurs at the same time and locality The sequestra often remain attached for a long time to the living bone at one spot.

4 SUPERFICIAL CARIES.—This occurs beneath the cartilage of a joint or the periosteum of the shaft The surface is covered by caseous debris. Beneath this is more or less osteosclerosis. At a little farther distance a zone of osteoporosis. It very seldom extends deeper than a quarter of an inch from the surface.

5 TUBERCULOUS PERIOSTITIS.—Commonest in the ribs and vertebræ Produces caries of the underlying bone, together with chronic abscesses spreading along the bones and tracking to a distance.

6 OSTEOMYELITIS (also epiphysitis).—Most often seen in the phalangeal, tarsal, and carpal bones.

TUBERCULOUS DACTYLITIS is common in children. A localized swelling occurs over a phalanx or metacarpal bone, in which tuberculous caseous processes expand the bone and break through the outer shell.

In adults, diffuse thickening from periostitis and the formation of central sequestra is commoner.

In either case neighbouring structures are affected by extension, viz.: (1) Joints, (2) Tendon sheaths; (3) Adjacent bones, especially in the wrist and ankle.

7. CARIES SICCA.—Is very rare, except in the shoulder-joint. Instead of soft tuberculous granulation tissue, firm fibrous tissue forms on the surface, and the bone steadily atrophies. It is accompanied by ankylosis with muscular atrophy.

8 DIFFUSE OSTEOSCLEROSIS.—This occurs for some distance along the shaft of a bone in the neighbourhood of an old tuberculous process The bone becomes dense and thick.

9. CHRONIC DEEP ABSCESS.—Usually situated near the epiphysial line It is lined by a thick pyogenic membrane and surrounded by a thickened mass of sclerosed bone. This thickening often extends up the shaft.

Distribution of Tuberculous Bone Lesions.—

CRANIAL BONES.—Rarely become carious, with external abscess

VERTEBRÆ.—Central osteomyelitis or superficial caries

RIBS.—Periostitis with abscess.

LONG BONES.—Epiphyses. caseation, necrosis, osteomyelitis, or chronic abscess—this is much the commonest situation. Diaphyses rarely the seat of periostitis, osteomyelitis, or chronic abscess.

PELVIS.—Near the sacro-iliac joint or any of the epiphyses, e.g , the crest of the ilium.

CARPAL, TARSAL, AND PHALANGEAL BONES.—Commonly affected by osteomyelitis, necrosis, or caries The os calcis, astragalus, scaphoid, or one phalanx, may be separately diseased, but usually more than one bone or joint is affected.

Signs.—

PAIN.—Generally in proportion to the depth of the lesion and its subjection to pressure. Pain of a dull aching character.

SWELLING.—(1) Of the bone, (2) Of the soft parts over it; (3) Abscess

ABSCESS.—An abscess forms without inflammatory phenomena, and in deep bone disease tracks to a distance.

FEVER.—Usually absent before the abscess breaks or becomes infected. Occasionally is well marked in purely tuberculous abscess of bones

JOINT SYMPTOMS.—Deformity, immobility, pain, etc

X RAYS.—Early radiographs are often negative, but later ones show marked decalcification, both in and around the lesion

Treatment.—

CONSTITUTIONAL TREATMENT, with rest to the affected part by immobilization. Usual to immobilize the limb with plaster-of-Paris. Passive hyperæmia in suitable cases.

OPERATIVE.—When the above has failed, or on the occurrence of an abscess, the part is laid open, the diseased part scraped away, sequestra are removed, and the cavity is rubbed with iodoform or filled with iodoform paste.

AMPUTATION is rarely required, and then generally in the case of a finger, hand, or foot, where the disease has spread to several bones and joints. Occasionally necessary in the foot when multiple sinuses are present and the majority of the tarsal bones are affected.

SYPHILIS OF BONES

Secondary Syphilis.—

SYMMETRICAL LOCALIZED PERIOSTITIS—hard nodes.—These occur most commonly on the tibia, associated with aching pain, worse when the part is warm, e.g., at night. It is a late secondary lesion, and may also occur in the tertiary stage

Tertiary Syphilis.—

1. GUMMATA.—These are usually subperiosteal.

THE CRANIAL BONES are the commonest site, especially the frontals and parietals. A firm nodular swelling first appears, which then softens and fluctuates. Bone beneath presents (1) CARIOUS DESTRUCTION, whereby a circular clean-punched hole is produced which very rarely perforates the skull; (2) AN OSTEOPLASTIC PERIOSTITIS at the margin, forming dense new bone; (3) AN EXTENSIVE SCLEROSIS, which may affect the whole vault of the skull or only that part under the gumma. When the gumma breaks and sepsis is admitted, a FOUL ULCER results and the bone becomes necrosed and black

BY AN EXTENSION OF THESE PROCESSES a large part of the cranium may be affected: (1) By worm-eaten erosion; (2) By heaping up of new bone; (3) By sclerosis; (4) By necrosis due either to sepsis or to a cutting off of the blood-supply by sclerosis. The sequestrum remains for years without separating

BY AN EXTENSION THROUGH THE SKULL, similar conditions may occur between the skull and dura, the deep surfaces of the bones being then affected.

THE PALATE AND MAXILLARY BONES, THE VOMER, ETHMOID AND NASAL BONES may be destroyed by submucous gummata, or by the extension of tertiary ulceration.

THE LONG BONES are rarely attacked by central gummata. In this case spontaneous fracture may occur, which is very slow in uniting.

2. OSTEOSCLEROSIS.—A diffuse osteoperiostitis may affect any of the bones, but especially the tibia, femur, humerus, radius, and ulna. Constant aching pain, worse at night, is complained of, and the subcutaneous parts of the bone become thickened. The X rays show irregular thickening under the periosteum of the whole shaft. The bone remains permanently hard and heavy, and necrosis may take place. (*See Fig.* 16, p. 53.)

Inherited Syphilis.—May affect the osseous system as follows:—

1. OSTEOCHONDRITIS OF THE NASAL SEPTUM, with necrosis of the cartilage—'snuffles' produced. Nose then becomes characteristically depressed.
2. EPIPHYSITIS within the first year. Epiphysis is broader than usual, of a yellow colour when sectioned. Usually symmetrical and associated with a periostitis extending along the shaft of the bone, giving the bone a fusiform enlargement, whereas in rickets the enlargement is irregular. Occasionally epiphysis is detached—pseudo-paralysis.
3. PARROT'S NODES ON THE SKULL.—Patches of local periostitis affecting the frontal bones—'hot cross bun' skull.
4. CRANIOTABES OF THE VAULT OF THE SKULL.—First six months—there is absorption of the bone. May be merely a constitutional effect as it also occurs in rickets.
5. SYMMETRICAL OVERGROWTH OF THE TIBIÆ.—Usually near puberty. Anteroposterior curvature of the tibia—'sabre tibia'. Tibia is sclerotic. May be associated with a symmetrical synovitis.

RICKETS

Aetiology.—Essentially a deficiency disease due to lack of vitamins A and D in the diet.

AGE.—Occurs in first and second years (*see also* LATE RICKETS, p. 224).

CLIMATE, etc.—Cold, damp, and dark encourage it Large cities have most of the cases.

DIGESTIVE DISTURBANCES.—Slight diarrhœa and vomiting often precede or accompany rickets.

ERRORS OF DIET are the most constant factors.

In BREAST-FED CHILDREN it is very rare, but occurs on prolonged lactation, or lactation during pregnancy.

IN HAND-FED CHILDREN it is common—either when fed on exclusively farinaceous food, or fed with cow's milk in a form in which the casein and fat are vomited.

THE DEFICIENCY OF PROTEIN, ANIMAL FAT WITH FAT-SOLUBLE VITAMIN and EARTHY PHOSPHATES in food, is the cause

DARK UNHYGIENIC DWELLINGS.

Symptoms and Signs : General.—

ANÆMIA, with marked deficiency in red cells

MUSCLES are flabby and weak.

BONES are all soft and inclined to bend

LIGAMENTS soften and stretch.

Tendency to PROFUSE SWEATING, especially over the forehead.

TEMPERATURE is subnormal.

Susceptible to gastro-intestinal and respiratory disturbances, e g., bronchitis and bronchopneumonia.

Protuberant abdomen, with large liver and spleen (these are probably cases complicated by congenital syphilis).

NERVOUS SYMPTOMS.—Convulsions—Laryngismus stridulus (laryngeal spasm often precedes a general convulsion)—Tetany (painful involuntary muscular contraction; especially affects the hands and feet; thumb is drawn into the palm, and fingers form cone-shaped hand)

GENERAL NUTRITION —Two types: (1) Thin, (2) Fat and flabby

Symptoms and Signs : Bones.—

CRANIUM.—

THIN PATCHES, from deficient inner table = craniotabes.

THICK BOSSES of red vascular bone on parietals, frontal, occipital.

Head is flattened from before backwards

FOREHEAD is broad, square, and projecting.

FOUR EMINENCES over the frontal and parietals are separated by a + groove.

FONTANELLES (especially the anterior) remain open up to the second or third year.

TEETH erupt late (eleventh or twelfth month). Come in irregular order. Fragile, deficient in enamel, and are shed early Notched with small segment of a large circle.

THORAX.—Transverse groove at the level of lower end of sternum. Vertical grooves at junction of ribs and cartilages. Prominent sternum. Beading of the ribs where they join the cartilages.

SPINE.—Long curve backwards (kyphosis). Often lateral curve (scoliosis). These often become permanent if they are not corrected before the rickets is cured.

Rickets—Symptoms and Signs—Bones, *continued.*

PELVIS.—Contracted and flattened from before backwards. Conjugate diameter, especially at brim, is diminished. Sacro-vertebral angle becomes acuter and more prominent Anterior superior iliac spines lie as far as or farther out than the crests of the ilia

ARM BONES.—Enlargement of lower radial epiphysis. Radius and ulna bend when child crawls

LEG BONES.—Enlargement of epiphyses, especially of lower tibial epiphysis. Bending outward and forward of lower third of tibia Bowing of the femur outwards and forwards Talipes valgus Talipes varus rarely, secondary to bowed tibiæ

Histology.—

EPIPHYSIAL CARTILAGE—Greater in circumference than normal More vascular. Thicker about quarter or half an inch, as compared with normal one-tenth

Zone of proliferating cartilage cells is thickened Cells are irregularly arranged. Cells are increased in proportion to matrix

Zone of calcified cartilage is thickened Calcification is very irregular

Line of ossification is uneven Islets of cartilage are left in the midst of ossified tissue

SHAFT.—Subperiosteal layer of bone is more vascular and spongy than normal. Hyperplasia and defective ossification occur in the surface of flat bones Compact bone and cancellous bone have vessels much larger and more numerous than normal, at the expense of the osseous tissue. Haversian canals are wider than normal

Results on the Bone as a Whole.—The bone is thicker, but weaker Contains only 40 per cent earthy matter, as compared with normal 60 per cent. Very liable to greenstick fractures Bends so that all natural curves are exaggerated.

ON RECOVERY—The spongy bone becomes somewhat denser than normal. The concavities of the curves are filled by buttresses of new bone, and thus the curves tend to be restored to the normal. This is in contrast to syphilis of bone where there is no buttressing of the bone. Premature ossification of the epiphysial cartilages leads to stunting of the growth

Varieties.—

FŒTAL RICKETS—Fractures of the bones, bending of the ribs, hyperplasia of cartilages, are sometimes found when the child is born.

SCURVY RICKETS—Often has a sudden onset, with pyrexia. Usually at 6–10 months Extreme tenderness of the limbs. Periosteal swellings of hæmorrhagic nature. Especially over the femur and tibia. The epiphyses may be detached, or spontaneous fractures occur Spongy gums. Ecchymoses and other hæmorrhages, e g , hæmaturia, epistaxis, or blood-stained diarrhœa, combined with other signs of rickets.

LATE RICKETS—Occurs about the age of puberty. Skull is not affected. Is similar to ordinary rickets but its effects are confined to the long bones, and the constitutional symptoms are much less marked. Its cause is unknown.

RENAL RICKETS.—Occurs in children. Usually between the ages of 12 and 14. Associated with the signs of renal insufficiency (often congenital lesions). Chronic interstitial nephritis. Urine copious, low specific gravity, blood-urea increased. Usually there is also overactivity of the parathyroids and an excessive calcium excretion. The bones become bent and also deformed at the ends by collapse of the metaphysis. When the hips are affected, it may be mistaken for tubercle. Due to a calcium/phosphorus imbalance and excessive calcium excretion in the urine. No treatment known. Any operation is likely to be fatal.

Treatment.—

THE DIET should contain fresh protein and fat in an assimilable form. Fresh milk, properly diluted and peptonized, if necessary, or mixed with lime-water. Raw meat juice, cream and eggs, custard puddings Also lime-juice, fruit, and vegetables in scurvy rickets.

SUNSHINE AND FRESH AIR.

DRUGS —These are only of secondary importance Cod-liver or halibut oil in thin children, and phosphate of iron

SURGICAL —In the acute stage of rickets, deformities should be prevented or corrected by splints, bandages, and manipulations, the child not being allowed to walk.

For deformities of the legs left after rickets has ceased, osteotomy will generally be required

OSTEOCHONDRITIS JUVENILIS DEFORMANS

(*Pseudocoxalgia Perthes', Legg's, or Calvé's Disease*).

Definition.—A flattening of the epiphysis of the head of the femur (*Fig.* 100) An affection of the epiphysis allied to Schlatter's disease of the tibial epiphysis and Köhler's disease of the tarsal scaphoid

Aetiology.—Generally occurs in healthy boys aged 5 to 10 years

Pathology.—Exact cause unknown Probably a low-grade pyogenic infection or the result of trauma Head of femur becomes fragmented and later 'mushroomed', with shortening of neck, and later neck becomes thickened and broad

Clinical Features.—Slight pain in the hip and associated limp Negligible wasting of the muscles Flexion practically full and extension full Abduction in the flexed position limited Inversion and eversion become limited as the head of the bone becomes flattened X-ray appearances vary with the stage of the disease No decalcification of the bones (cf. tuberculous hip). The head of the femur is dense and flattened and later becomes 'fragmented' in appearance, and the neck of the femur thickened and broad.

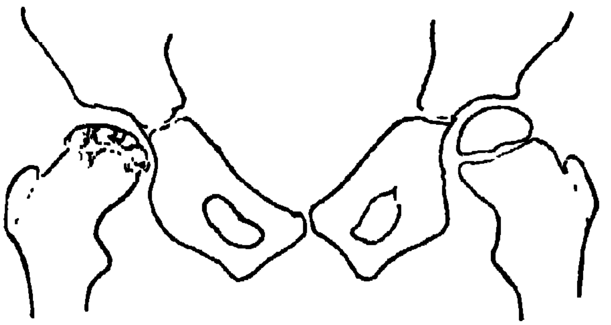

Fig. 100.—Perthes' disease.

Osteochondritis Juvenilis Deformans, *continued.*

Treatment.—Rest in recumbency with traction on the affected femur is necessary until X rays show that the head has re-formed. This may need twelve months of treatment. Adequate early treatment prevents osteo-arthritic changes developing in later years

ACHONDROPLASIA

Definition.—A defective growth of all the bones which develop from cartilage. It differs from rickets in not manifesting its results until some years after birth. It is often hereditary

Stunting of the Long Bones.—The growth of the shaft of the bones from the epiphysial cartilage is very slight, so that the limbs grow in thickness but not in length The adult is a dwarf

Changes in the Head.—The vault of the skull, which grows from membrane, is normal, whilst the base of the cranium and face are stunted. Mental condition is normal

Hands.—The stumpy, thick fingers cannot touch one another at their tips, but diverge like the spokes of a wheel

OSTEOMALACIA

Definition.—An absorption of the hard parts of the bones, resulting in bending and fractures

Aetiology.—In over 90 per cent it affects women, and usually begins during pregnancy.

Pathology.—The compact parts of the bone are replaced by a fibro-cellular tissue, whilst very vascular pulpy marrow fills the medullary cavity The earthy salts are reduced to a sixth of their normal proportion. Possibly the absorption of the bony salts may be due to the over-production of an internal secretion by the ovaries, together with lack of sunlight.

Symptoms.—Severe deep-seated pain in the spine, pelvis, and leg bones. Emaciation and exhaustion Later the limbs become bent or broken, and the pelvis is pressed into a triradiate shape which makes parturition impossible

Treatment.—Oöphorectomy has no influence on the disease. Diet rich in calcium and vitamin D.

GENERALIZED OSTEITIS FIBROSA

(*Fibrocystic Disease, Von Recklinghausen's Disease*)

Definition.—A disease of middle age, in which a development of fibrous tissue and cysts occurs in the shafts of the long bones, females being more often affected than males.

Symptoms.—Pain of a dull aching character is common, even before fracture has taken place. General lassitude and debility are present in advanced cases. Commonly only one or two bones are affected. Sometimes many of the long bones and phalanges, together with the skull and pelvis, are the seat of disease.

Three abnormal tissues may take the place of the bone-marrow or of the cancellous tissue, and these may be present alone or together in varying proportions:—

1. Cysts with a simple lining. These may develop after a fracture, or they may be the cause of a spontaneous fracture.
2. Large masses of white fibrous tissue which cuts like a turnip.
3. Collections of cellular material in which are contained many multinucleated giant cells.

The development of fibrocystic tissue in the centre of the bones leads to bending or to fracture. The shaft of the humerus and the upper end of the femur are the commonest sites for these manifestations. If untreated the fractures heal very badly and the deformities progress.

Urinary symptoms: Urinary calculi are common, in the kidneys or bladder.

Pathology.—Associated with and probably resulting from a parathyroid excess. There is usually present a parathyroid tumour. There is a high blood-calcium and a high calcium excretion, 16–18 mg./100 c.c. Blood-phosphorus is normal.

Treatment.—The association of generalized fibrocystic bone disease with high blood-calcium justifies exploration for a parathyroid tumour, which may be embedded in the thyroid gland or hidden in some recess behind the thyroid or behind the sternum

LOCAL TREATMENT OF THE BONE LESIONS.—*See below.*

LOCALIZED FIBROCYSTIC DISEASE

Special Features.—In this disease only one or two bones are affected. The patient is usually young, about 15 to 25 A localized cyst is associated with a spontaneous fracture. There is no change in the calcium metabolism and no parathyroid derangement

Treatment.—Expose the site of the cyst, open fully and scrape out the cavity. Splint efficiently so as to prevent deformity. The most efficient kind of splint is an internal bone-graft Cure often results following spontaneous fracture

OSTEITIS DEFORMANS

(*Paget's Disease of Bone*)

Definition.—A disease consisting in late overgrowth of the long bones, with thickening and bending.

Pathology.—The bones become softened, thickened, and lengthened. The natural curves are exaggerated by bending. The cause is quite unknown In a few cases multiple sarcomata have grown in the affected bones. There is atrophy of the compact tissue and absorption of the calcium salts and a deposition of subperiosteal new bone

Symptoms and Course.—Severe dull aching pains come and go, and are usually regarded as rheumatic. The onset is very insidious, and the patients live to an old age.

Deformities.—

The CRANIUM becomes thickened and enlarged by an eccentric hypertrophy, the face remaining unaffected.

THE SPINE assumes a long kyphotic curve, with much pain and stiffness.

THE LEG BONES become bowed forwards and outwards.

Treatment.—Linear osteotomy for relief of pain.

ACROMEGALY

Definition.—A bony overgrowth affecting chiefly the jaw, hands, and feet, and leading to gigantism.

Aetiology.—Men and women are equally affected, and the disease begins in young adult life

Pathology.—Always associated with overgrowth of anterior half of pituitary body, which may ultimately lead to death by cerebral compression. Bony changes are merely those of overgrowth

Symptoms.—Pain is indefinite and intermittent. Headache and lassitude. Some abeyance of the sexual functions, shown by amenorrhœa in women and impotence in men Appetite and thirst are excessive.

Signs.—The hands and feet enlarge, but more in length than breadth, the fingers becoming spatula-shaped The jaws, especially the lower, become large, and the lips thick. Gigantism is not always present, but nearly all giants are examples of acromegaly

NEW GROWTHS OF BONES

Innocent Tumours.—

FIBROMA (periosteal)
LIPOMA (periosteal)
CHONDROMA
OSTEOMA (exostosis)

ANEURYSM BY ANASTOMOSIS —A rare condition, found usually in the cranial bones, producing thinning of the bone over a pulsating tumour.

BLOOD CYSTS.—Found in the cancellous ends of the long bones, probably the remains of myeloid sarcomata.

SIMPLE CYSTS.—Fibrocystic disease occurs in the shafts of the long bones, especially in the humerus or femur It causes atrophy of the bone with bending or fracture.

HYDATID CYSTS.—May occur in the long bones or the pelvic bones, arising in the medullary cavity without the formation of any parent cyst, the daughter cysts lying free. A chronic thickening of the bone occurs, later a spontaneous fracture

OSTEOCLASTOMA.—The commonest growth It occurs in the lower ends of the femur and radius, and the upper ends of the tibia and humerus (i.e., the ends, in which the most active growth takes place); also in other epiphyses, the lower jaw, the diploë of the skull, or as an epulis. It is generally innocent, rarely causing metastases, and not recurring after local removal. It is extremely vascular, the blood spaces often breaking down to form hæmorrhagic cysts It often pulsates A bony skeleton pervades the growth. Egg-shell crackling is common (*Fig.* 101.)

Treatment.—X rays and radium have a beneficial effect, but this is only temporary Small early growths are treated by curetting and swabbing with pure carbolic acid. Larger growths, by excision of part of the articular end, using a bone-graft for reconstruction.

MALIGNANT TUMOURS

May be: (1) Primary; (2) Secondary

Primary Malignant Tumours.—(1) Sarcomata; (2) Ewing's tumour or sarcoma; (3) Multiple myelomata.

. SARCOMATA.—May be: (*a*) Parosteal. (*b*) Periosteal. (*c*) Endosteal.

a. PAROSTEAL.—Spindle-cell tumours at insertion of muscles and tendons. Local and free excision. Improper excision will result in recurrence with increased malignancy.

b. PERIOSTEAL.—Very malignant—puberty and adolescence—ends of long bones, usually lower end of femur and upper end of tibia (*Fig*. 102). Leg affected 4–5 times more often than the arm. Rapid growth and pain an early feature as periosteum is stretched. Affected area has distended veins over it and is usually spindle shaped. Later the tumour may become pulsatile. Soft tissues invaded later. Dissemination is early and rapid, by the blood-stream. Secondary deposits in the lungs.

X-ray Appearances.—(1) Rapidly growing tumours show bone destruction (osteolytic type). (2) Less rapidly growing tumours produce subperiosteal new bone giving the characteristic 'sun-ray' spicule formation.

Diagnosis.—Either very easy or difficult. Slowly growing sarcomata may be confused with a syphilitic process If Wassermann reaction is negative, diagnostic deep X-ray therapy is indicated; no effect on inflammatory processes in bone, but causes bone formation in a sarcoma Biopsy is contra-indicated

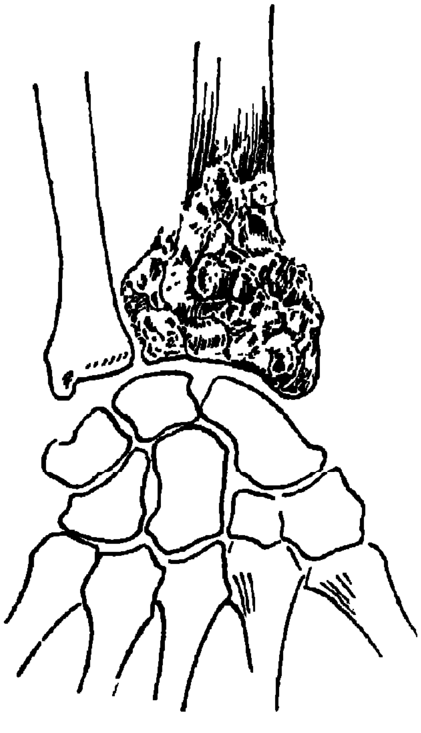

Fig. 101.—Osteoclastoma of lower end of radius.

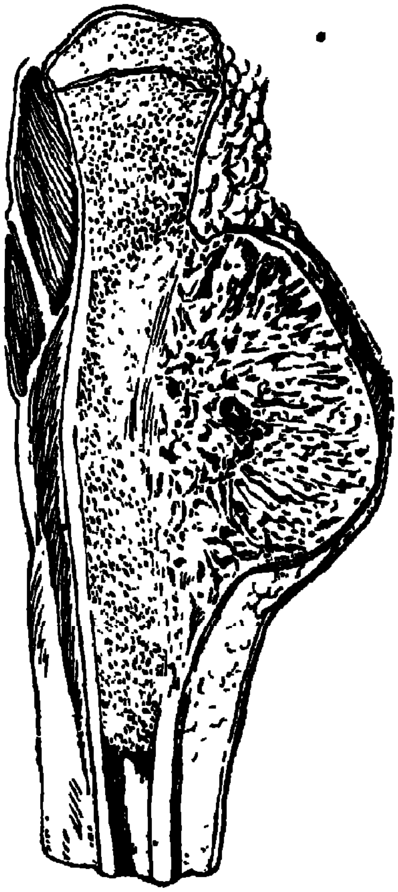

Fig. 102.—Periosteal sarcoma of tibia.

Primary Malignant Tumours—Sarcoma, ***continued***

c ENDOSTEAL.—Round or spindle shaped—usually medulla of a long bone Pain due to rapid growth and destruction rather than expansion of the bone. Spontaneous fracture may occur May be confused on radiography in early stages with a myeloma Destruction of compact bone with little sclerosis and periostitis

Treatment.—

1. Amputation. High above the growth, or, if not possible, above the growth followed by deep X-ray therapy Always X-ray chest to exclude secondary deposits
2. Injection of Coley's fluid (mixed toxins of streptococcus of erysipelas and *B prodigiosus*) Doubtful value now being abandoned
3. Deep X-ray therapy

Prognosis —Poor. Of 650 cases, only 17 cured

2. EWING'S SARCOMA —Rare tumour affecting the middle of the shafts of long bones. Signs resemble a subacute osteomyelitis—pain, local tenderness, and slight pyrexia X-ray shows bone destruction with some sclerosis around and the laying down of subperiosteal new bone in definite layers parallel to the shaft Prognosis is poor. Deep X-ray therapy causes rapid retrogression of the growth, but local recurrence is usually the rule Secondary deposits are less radiosensitive, they occur in glands and the viscera

3 MULTIPLE MYELOMA Age 40 50 Multiple growths in the marrow —usually spine and flat bones— with destruction of cortical and compact bone Bence-Jones proteose in urine X-ray resembles multiple secondary deposits Treatment of no avail

Secondary Malignant Tumours.—

CARCINOMA —From primary sites in breast, bronchus, thyroid, prostate, and kidney Cause pain and spontaneous fracture Union may occur Secondary deposits from prostate are osteosclerotic

SARCOMA.—Spontaneous fractures occur never unite

Chapter XXII

INJURIES OF JOINTS

SPRAINS

Definition.—Subcutaneous traumatic injury of soft parts connected with a joint, i.e., of muscles, tendons, and ligaments

Varieties.—

Sprain Without Swelling.—Otherwise known as a strain. Parts have been stretched and not torn

Simple Uncomplicated Sprain.—Muscles, tendons, or ligaments torn.

Sprain Complicated by a Fracture

Sprain Complicated by a Nerve Injury

Symptoms.—

PAIN.—Localized—due to direct injury. Referred—due to nerve lesion.

SWELLING.—Immediate—due to hæmorrhage, may occur at a distance from injury, e.g., in ham from sprain in thigh. Late—due to inflammatory effusion

NUMBNESS.—Temporary—due to nerve shock. Lasting more than twelve hours—due to nerve lesion

Diagnosis.—

1. Fracture must be determined or excluded by X rays
2. Gross Nerve Lesion.—Distant pain, numbness or paralysis
3. Dislocation.—Alteration of relations of bony points

Treatment.—

STRAINS, OR SPRAINS WITHOUT SWELLING.—

Injection of Local Anæsthetic into the affected area, firm bandaging, and encouraging early active movements

Massage begun with twenty-four hours.

SIMPLE SPRAINS WITH IMMEDIATE SWELLING.—

Rest, with hot fomentations until increase of swelling ceases. Hot lotio plumbi c opio

Elastic Pressure with crêpe bandage or strapping

Voluntary Movements as soon as can be used without pain

If an important ligament has been torn, e.g., the lateral ligament of knee or ankle, then efficient splinting should be adopted, so that no strain can be put on the torn ligament for four to six weeks

SIMPLE SPRAINS WITH LATE SWELLING.—

Elevate the limb till swelling disappears. If necessary aspirate the joint. Put on a firm covering of Unna's paste or Elastoplast

TREATMENT OF SPRAINS WITH A FRACTURE.—If only fissured, and not complete fracture, treat as above

If fracture has detached piece of bone, put up in a plaster case with early active movements of the nearby joints.

Sprains—Treatment, ***continued.***

TREATMENT OF SPRAINS ASSOCIATED WITH GROSS NERVE LESIONS.—

For painful nerve lesions: Rest, and not massage.

For numbness: Massage, galvanism, or operation.

Remote Results of Sprains.—

PREVENTABLE —

PAIN and STIFFNESS OF JOINTS —The late results of immobilization and want of active movements

MUSCULAR WASTING AND RELAXATION OF JOINT.—Should be prevented by massage and electricity

DEFORMITY arises from a fracture which has been overlooked.

UNAVOIDABLE sometimes —Osteo-arthritis—Local paralysis—Myositis ossificans.

MYOSITIS OSSIFICANS —Arises in torn muscles Nearly always associated with unwise or ill-timed passive movements Slow development. Not painful Swelling of affected muscles Stiffness and difficulty in movement

TREATMENT —Prolonged rest (six months) Mass diminishes, but does not disappear.

DIAGNOSIS —From sarcoma or periostitis

X rays show mass in muscle away from bone Absence of pain or heat.

DISLOCATIONS

Causes.—

1. TRAUMATISM —
2. CONGENITAL CONDITIONS —

CAUSES (theoretical) —Malposition or injury in utero Failure of one of the joint surfaces to develop, e g, rim of the acetabulum, iliac portion of the acetabulum, head of the femur Fœtal disease of the joint or muscles

JOINTS AFFECTED —Hip (common), jaw, shoulder, wrist, and patella (rare)

3 PATHOLOGICAL CONDITIONS —May be·—

1 DISTENSION of capsule by exudation.
2 DESTRUCTION of either articular surface by disease.
3. MUSCULAR TRACTION after the destruction of ligaments.

Any of these may occur in Acute septic arthritis (especially 1), tuberculous disease (especially 2 and 3), osteo-arthritis; Charcot's disease.

TRAUMATIC DISLOCATIONS

Causes.—

PREDISPOSING —Shallow articular cavity. Lax muscles and ligaments Adult age and male sex

EXCITING.—Violence—generally indirect Muscular action in lower jaw, patella, or humerus—rare except after previous dislocations.

Varieties.—

COMPLETE.—Articular surfaces are completely separated.

INCOMPLETE (subluxation) —Articular surfaces only partly separated.

COMPOUND.—External wound communicates with joint cavity.

COMPLICATED —Associated with injury of vessels nerves or viscera.

Signs.—

LOCAL CONTUSION OF PARTS.—Pain—Swelling—Bruising.

RESTRICTED MOBILITY.

DEFORMITY.—Articular surfaces felt in abnormal position. Altered relation of bony points. Altered axes of bones related to the joint.

Anatomy of Dislocation.—

OF RECENT INJURIES.—

Ligaments are torn, especially the capsular ligament.

Muscles torn or stretched.

Tendons stretched, torn, or hitched round dislocated surfaces.

Vessels and Nerves contused or torn

Articular Cartilage fissured or torn

Bony Points often fractured

Articular Cavity and surrounding parts are filled with blood

OF OLD UNREDUCED DISLOCATION —

Articular Cavity becomes filled with fibrous tissue

Head of Bone loses its articular cartilage, becomes buried in dense adhesions or surrounded by new false joint

Muscles become adaptively shortened

Tendons acquire new attachments. Muscles, tendons, and ligaments are matted in fibrous tissue.

Bone on which movable bone rests atrophies at point of contact. Is heaped up by periostitis round the point of contact

If the dislocation is kept at rest for several weeks, Fibrous Union takes place.

If the dislocated limb is not kept at rest, a False Joint is formed.

Treatment of Dislocations.—

REDUCTION by —

Manipulation, to make the head of the bone retrace its way through the hole in the capsule

Traction, to overcome muscular tension Preferably under an anæsthetic Rarely with the aid of pulleys

Operation —When the above measures have failed When fracture which prevents retention exists, e g , forward dislocation of elbow with fractured olecranon When fracture which prevents reduction exists, e g , dislocated shoulder and fractured neck of humerus When complicated by ruptured vessels or nerves.

RETENTION in position of rest.—Rest for one to three weeks according to the size of the joint. Active movements of neighbouring joints and faradism to the immobilized muscles. Massage and passive movement at the end of first week Active movements in addition at the end of first, second, or third week

DIFFICULTIES IN REDUCTION are caused by. Torn capsule embracing neck of the bone—Tonic contraction of muscles—Interlocking of bony points—Tendons and muscles catching round the neck of displaced bone

Treatment of Old Unreduced Dislocations.—

REDUCTION should be attempted up to the end of the third month. Later than four or six weeks it is unlikely to succeed.

Dangers.—To main vessels and nerves Fracture of the ends of the bone. Tearing of the soft parts.

Dislocations—Treatment of Old Unreduced Dislocations, ***continued.***

MASSAGE AND SYSTEMATIC MOVEMENTS are the best treatment: When some mobility exists—Where there is slight pain—In elderly patients.

OPEN OPERATION is indicated: When no movement is possible without pain—Especially in joints of the upper limb—Especially in young and working men.

ATTEMPT AT REPOSITION by division of tendons and adhesions.—Generally impossible because articular cavity no longer exists.

EXCISION OF THE JOINT —Especially useful in the shoulder or elbow, where mobility is of more importance than stability

SPECIAL DISLOCATIONS

LOWER JAW

Simple Dislocation.—

DIRECTION —FORWARDS, generally bilateral

CAUSES.—Yawning—Gags and dental operations

SIGNS —Mouth cannot be shut In unilateral cases the chin is displaced laterally A depression occurs in front of the ear

TREATMENT —Pressure downwards and backwards on the lower molars by the guarded thumbs

Internal Derangements.—In persons over middle age Painful clicking and snapping in the joint Caused by tear or deformity of the inter-articular cartilage

TREATMENT —By removal of cartilage

CLAVICLE

Sternal End.—

VARIETIES —

FORWARD —The bone lies on the manubrium sterni

BACKWARD —Symptoms occur from pressure on the trachea, œsophagus, and vessels

UPWARDS —Very rare

TREATMENT —Draw shoulders back and fix with an 8 bandage

Acromial End.—

VARIETIES —UPWARDS or DOWNWARDS

TREATMENT —Easy to reduce, difficult to maintain Bandage over the shoulder and under the flexed elbow Unreduced dislocation is often of little importance. If it gives rise to pain and stiffness, treat by an open operation, maintaining the reduction by using strips of fascia lata. If conoid and trapezoid ligaments affected, then fixation of the clavicle to the coracoid by means of a screw.

SHOULDER

Primary Dislocation.—

PREDISPOSING causes —Shallowness of glenoid cavity. Laxity of capsule Freedom of movements.

EXCITING CAUSES.—Falls on outstretched hand or elbow. Violent muscular exertion in cases that have been dislocated before.

MECHANISM.—Head of humerus tears through capsule in front or below. Subsequently displaced forwards or backwards.

VARIETIES and anatomy of each:—

1. Subcoracoid.—Head lies below coracoid process upon the neck of the scapula (*Fig.* 103). Tendon of subscapularis is torn or stretched over the neck of the humerus Supraspinatus, infraspinatus, and teres minor are either tightly stretched, producing external rotation, or torn (sometimes with great tuberosity), with internal rotation.
2. Subclavicular (rare).—Exaggeration of the subcoracoid variety. The great tuberosity and coracoid process are fractured or their muscles are torn. Head of bone lies below clavicle.
3. Subglenoid (rare) —Head of bone lies on axillary border of scapula below the glenoid cavity. Muscles attached to tuberosities are torn. Axillary vessels compressed
4. Subspinous Dislocation.—Head rests on the posterior aspect of the neck of the scapula Subscapularis muscle is torn.
5. Supracoracoid (very rare).—Acromion (and possibly coracoid) fractured. Head of the bone tears through upper part of capsule.
6. Luxatio Erecta (very rare).—Head of bone lies below glenoid cavity. Arm is stiffly held above the head Rotator muscles are intact.

SIGNS OF DISLOCATION —

Local Contusion.

Restricted Mobility.

Flattened Outer Border of Shoulder.—Straight edge can touch acromion and external condyle

Head of Bone is felt in an abnormal position.

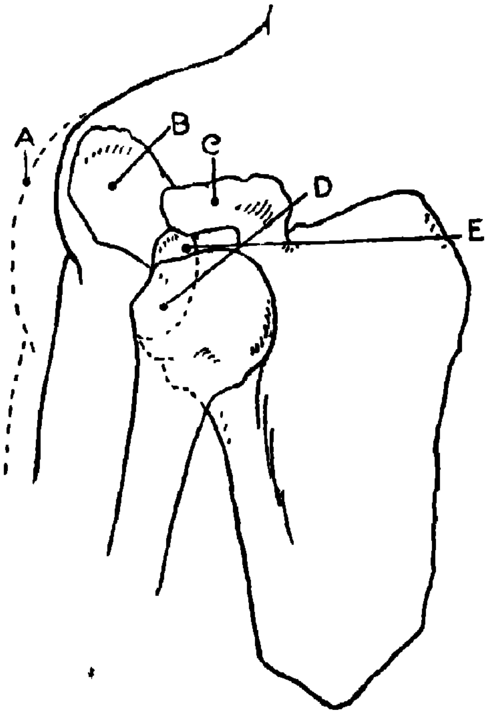

Fig. 103.—Subcoracoid dislocation of shoulder. A, Outline of normal shoulder; B Acromion, C, Coracoid; D Head of humerus; E, Glenoid

Special Dislocations—Shoulder—Signs, ***continued.***

Elbow Displaced from the side, and cannot touch the chest wall when the hand is on the opposite shoulder.

Vertical Measurement round the axilla is increased.

Anterior or Posterior Fold of the axilla is lowered.

Measurements.—From acromion to external condyle is customarily unaltered, but there may be slight shortening, especially in the subclavicular. In the subglenoid variety there is an inch lengthening.

SPECIAL SIGNS of the different varieties:—

Subcoracoid.—Head felt below outer end of the clavicle. Elbow is directed outwards and backwards. Shortening absent or very slight.

Subclavicular.—Head felt below middle of the clavicle. Shortening may be present. Elbow is far from the side.

Subglenoid.—Head is felt in the axilla Signs of pressure on vessels and nerves Marked lengthening of the arm Flexion of elbow from tension on biceps.

Subspinous.—Head of the bone felt behind, below the acromion. Elbow displaced forwards Arm rotated inwards

TREATMENT OF SHOULDER DISLOCATIONS.—

1. **Manipulation** under anæsthetic.

Kocher's Method.—Elbow is held to the side Hand is brought forwards and outwards, so as to rotate the humerus externally and relax the

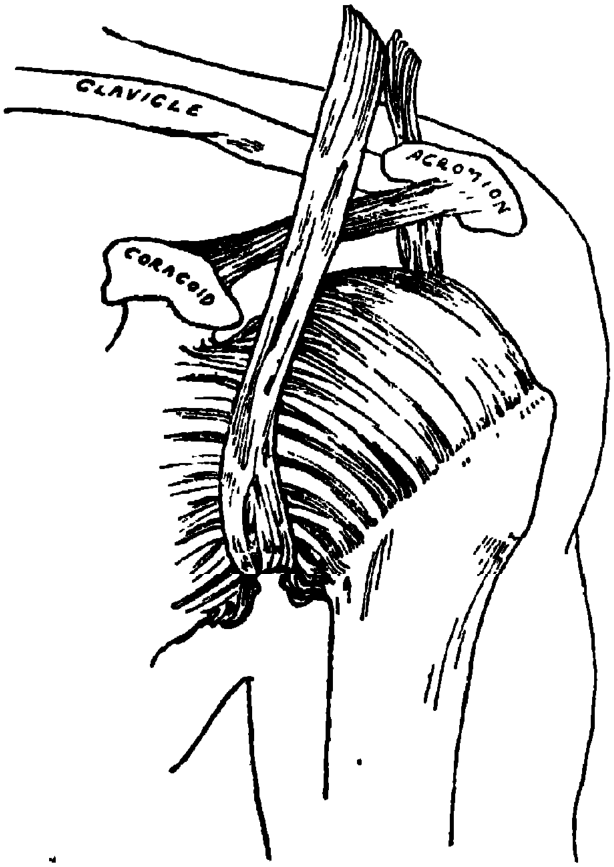

Fig. 104.—Sling operation for shoulder dislocation.

external rotators. Elbow is adducted to the mid-line—this makes the margins of the gap in the capsule tense. Elbow is raised, so as to slacken upper margin of the rent and keep lower tense. Hand is placed on the opposite shoulder, i.e., arm is rotated inwards, to make the head of the humerus slip into the capsule. Elbow lowered.

2. EXTENSION.—Traction on the hand whilst counter-extension is made in the axilla by the heel of the operator's unbooted foot.

Recurrent Dislocation.—The shoulder after simple dislocation is very liable to recurrence of the displacement owing to laxity and weakness of the capsule and shoulder muscles

Essential pathology of recurrent dislocation of the shoulder-joint is: (1) Injury and displacement or detachment of a portion of the glenoid labrum. (2) Injury to the posterolateral sector of the head of the humerus.

TREATMENT —(1) By forming a muscle sling (*Fig* 104). The anterior and posterior borders of the deltoid are exposed, a strip from the posterior border is brought under the joint and sewn in front. (2) A similar exposure, but using a long strip of fascia lata The ends of the latter are fastened over the acromion (3) Nichols's operation Dividing the tendon of the biceps at the bicipital groove and passing the tendon through a drill hole in the head of the humerus and then suturing it to itself (4) Bankhart's operation Gives the best results. The tear in the glenoid labrum is exposed and the detached portion sutured into position.

ELBOW

Ulna and Radius.—

VARIETIES.—BACKWARDS—FORWARDS—LATERAL.

BACKWARD DISLOCATION (the common variety).—The coronoid process is often broken The distance from the acromion to the condyles is unaltered The distance of the olecranon from the condyles is increased. The distance of the condyles from the styloid processes is less. The olecranon is unduly prominent The arm is held flexed.

FORWARD DISLOCATION (rare, except as a complication of fractured olecranon).—Forearm is markedly lengthened. Condyles are unduly prominent The olecranon is obscured

LATERAL DISLOCATIONS —Very rare and usually incomplete.

TREATMENT.—Flexion of the forearm over the operator's knee placed in the bend of the elbow

The Ulna Alone (very rare) —BACKWARD

The Head of the Radius.—

VARIETIES —COMPLETE—FORWARD (the common variety)—BACKWARD or OUTWARD (very rare)—INCOMPLETE—SUBLUXATION.

FORWARD DISLOCATION.—Prominence of the head of the radius in front of elbow. Hollow behind the external condyle Flexion at the elbow is incomplete. Supination is defective. Easy to replace, difficult to retain owing to the rupture of the orbicular ligament

Anterior dislocation of head of radius may be associated with a fracture of the upper third of the ulna—i.e , Monteggia fracture.

Treatment.—Flex with a pad in the elbow. Keep flexed for two or three weeks. Massage and passive movements at end of seven days. In late cases, the orbicular ligament must be replaced by a sling of fascia or tendon tying the radius down to the olecranon.

Special Dislocations—Elbow—Head of the Radius, *continued*

SUBLUXATION ('pulled elbow').—The radial head is pulled downwards out of the orbicular ligament. The arm is fixed in a partly flexed position. Results in children from their being lifted up by one hand *Treatment.*—By complete flexion and then extension.

WRIST

The Complete Wrist.—

VARIETIES.—BACKWARDS (rare)—FORWARDS (very rare).

SIGNS.—Both styloid processes project prominently, but their relation to each other is normal. The carpal bones from a mass at the back of the joint, and their relations to the styloid processes are altered.

The Semilunar Bone.—More common than that of the wrist, usually following forced dorsiflexion Displaced forwards and rotated Grasping movements weak and pain felt in distribution of the median nerve.

TREATMENT.—Continuous traction for 10 minutes. In old unreduced dislocations excision should be performed

FIRST PHALANX OF THUMB

DIRECTION.—BACKWARDS

ANATOMY.—The head of the metacarpal becomes entangled by. (1) The tendon of the long flexor, (2) The glenoid ligament, i.e, the capsule of the joint, (3) The short muscles attached to the sesamoid bones.

REDUCTION.—By traction followed by flexion Is often very difficult. In this case open the joint behind and cut the glenoid ligament at its attachment to the base of the phalanx

HIP

Traumatic Dislocation.—

ANATOMICAL FACTORS.—

The ilio-femoral ligament is the strongest part of the capsule. It is usually unruptured, the dislocation in this case being one of the regular varieties. If it is torn, the dislocation will be irregular.

The tendon of the obturator internus runs over the neck of the femur behind. In backward dislocations its integrity will prevent the femoral head going up to the dorsum ilii.

The rim of the acetabulum, especially above and behind, may be chipped off; this makes retention after reduction very difficult.

The capsule is usually torn below or behind at its weakest place.

VARIETIES.—BACKWARD: (1) On to the dorsum ilii; (2) On to the sciatic notch. FORWARD: (3) On to the obturator foramen, (4) On to the pubis.

DORSAL DISLOCATION.—Head of femur lies on the dorsum ilii, and can be felt in the buttock. The obturator internus is ruptured in most cases. The short rotator muscles are lacerated. The trochanter lies well above Nélaton's line, and approximated to the anterior superior iliac spine. The leg is shortened two to three inches The iliotibial band is relaxed. The leg is flexed, adducted, and inverted. The femur crosses the lower third of the opposite thigh. The toe rests on the opposite instep. A hollow exists in Scarpa's triangle.

SCIATIC DISLOCATION.—Similar to the above, except in the following: The obturator internus tendon is intact and lies over the neck of the femur, holding it down in the sciatic notch. Shortening amounts only to one inch or less. The axis of the femur crosses the opposite knee The great toe rests on the dorsum of the opposite great toe.

TREATMENT OF THE BACKWARD DISLOCATIONS.—

MANIPULATION.—Flex the knee and thigh in position of adduction. Abduct the thigh and evert simultaneously. Bring the leg down straight "Lift up, bend out, roll out."

DIRECT TRACTION of the thigh forwards in a line at right angles to the body.

OBTURATOR DISLOCATION.—The head of the bone lies on the obturator externus in the obturator foramen. The adductor muscles are lacerated. The trochanter is obscured, the iliotibial band is tense. The leg is lengthened, the toes point forwards and outwards. Flexion, abduction, and rotation outwards are well marked. The head of the femur is felt in the perineum The capsule is torn in its lower part. Pain referred to the distribution of the obturator nerve.

PUBIC DISLOCATION —Similar to the above except· The femoral head is felt under Poupart's ligament. The leg is shortened about one inch. Abduction and eversion are more marked, the toes pointing outwards

TREATMENT OF FORWARD DISLOCATIONS.—Thigh is flexed in a position of abduction Adduct the thigh and then invert it Bring the thigh down straight "Lift up, bend in, roll in"

Congenital Dislocation (*Fig.* 105) —

SYMPTOMS —

Child begins to walk late. Deformity is often noticed only after walking has begun. Gait is waddling and rolling from side to side. Girls more frequently than boys. May have associated developmental anomalies.

Buttocks are very prominent behind. Hips are very broad laterally. Belly is very protuberant

Marked lordosis is always present Scoliosis is evident in unilateral cases.

Heel is not brought to the ground on affected side

Fig. 105.—Congenital dislocation of hip after correction in plaster case.

Congenital Dislocation of the Hip, *continued.*

POSITION OF LEG.—Thigh is flexed (by traction of iliopsoas), adducted (by traction of adductors), slightly inverted (by traction of the anterior part of the capsule).

Shortening of one to three inches.

Trochanter is much above Nélaton's line, and may be at the level of iliac crest; it is farther from the mid-line than normal.

A gap occurs in the perineum between the thighs

TRENDELENBURG'S SIGN (also given in paralysis affecting the glutei groups) —When walking and stepping on the affected leg, the opposite side of the pelvis drops, instead of being raised

POSITION OF THE PELVIS —Flexed on the thigh by traction on iliopsoas; consequent great lordosis Often tilted laterally in unilateral cases.

MOBILITY.—Free mobility, except in abduction. Limb can be drawn down to its full length by traction in the flexed position, especially in early cases.

PAIN.—Usually absent. Fatigue and aching after walking are noticed in late cases.

Passive movements are painless.

IN UNILATERAL CASES —Waddling and rolling are not so marked Lameness is much less conspicuous Scoliosis of lumbar region occurs, with convexity on lame side There is a difference of one to three inches in the length of the two legs

X RAYS —Trochanter is on a level with anterior superior iliac spine. Acetabulum is deficient in its iliac segment Head of the femur is absent or malformed. The angle between the neck and shaft is more open than normal (coxa valga). A ridge of new bone may jut out from the dorsum ilii above the femur, representing the false acetabulum

PATHOLOGICAL ANATOMY.—

Acetabulum.—Iliac segment is deficient Cartilaginous rim is absent. It consists of a triangular shallow depression at union of pubis and ischium (*Fig* 106).

False Acetabulum.—In old cases that have walked, a rim of new bone may be present on the dorsum ilii, forming a new acetabulum

Head of the Femur is stunted or absent The neck is short and anteverted.

Capsule is lengthened and thickened It presents a well-marked hourglass contraction, which after 5 or 6 years is too tight and too strong to permit the head of the bone being pushed through it into its socket (*Fig* 107).

Ligamentum Teres is long and band-shaped, or absent.

DIAGNOSIS from:—

Coxa Vara.—Comes on in childhood or puberty after child has walked well. Eversion. Limb cannot be lengthened by traction. Radiograph shows deformity of neck.

Tuberculous Hip.—Late onset of symptoms. Pain is much more marked. Rigidity of the joint. Limb cannot be pulled down.

Traumatic Dislocation.—History of difficult labour. Limb cannot be pulled down. Radiograph shows normal bones.

TREATMENT.—

In Early Cases (up to 6 Years).—

Lorenz' Bloodless Operation (under general anæsthesia).—Limb is pulled down, flexed, abducted, and everted. Adductors are ruptured by kneading Fixed by plaster bandage, including pelvis and thigh: position of extreme abduction and slight eversion (*Fig.* 105). Retained for twelve weeks. Fixed again with less abduction for six months.

In Older Cases.—Open operation. An incision along crest of ilium and down along line of sartorious. All tissues separated from outer surface of ilium, and acetabulum exposed Capsule opened and hour-glass constriction divided (*Fig* 107). Head of bone levered into position. If the socket is too shallow to retain the head, then the upper margin of the acetabulum must be increased by turning down a bone flap from the outer surface of the ilium or wedging in a bone-graft.

In Adult Cases —An osteotomy just below the small trochanter. The shaft of the femur is fully abducted This causes the pelvis to be tilted down on the affected side, thus making the limb so much longer. The pelvis is supported on the crooked femur instead of hanging on the capsule. This cures the pain.

PATELLA

Primary Dislocation.—

VARIETIES —Outwards, Inwards, or Vertical Rotation

Genu valgum or varum are predisposing causes Muscular violence or direct violence may cause it

Accompanied by a rupture of a part of the extension of the quadriceps aponeurosis

TREATMENT.—Flex the thigh, extend the knee, and press into place

Recurrent Dislocation.—This is nearly always outwards, and is associated with genu valgum It is treated by operative correction of the genu valgum, in addition to which one of the muscles on the inner side of the knee, e g., the gracilis or semitendinosus, is attached to the inner border of the patella.

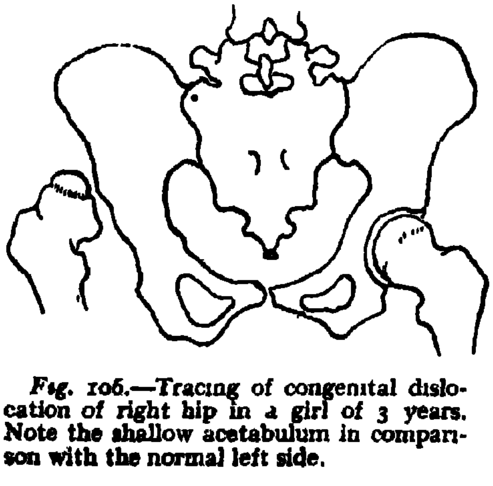

Fig. 106.—Tracing of congenital dislocation of right hip in a girl of 3 years. Note the shallow acetabulum in comparison with the normal left side.

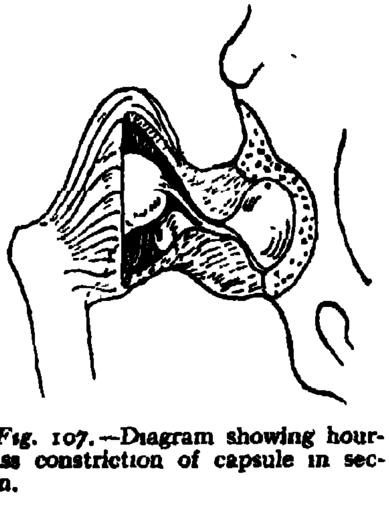

Fig. 107.—Diagram showing hour-glass constriction of capsule in section.

KNEE

DISLOCATION OF KNEE-JOINT

VARIETIES.—Forwards, Backwards, and Towards Either Side. May be associated with rotation of the leg bones on the femur. Dislocation of the leg to the lateral side is the commonest.

CAUSE.—Always due to great direct violence

ANATOMY.—Complete dislocation always involves tearing of the ligaments, notably the cruciate and collateral ligaments, and also rupture of the popliteus and both heads of gastrocnemius muscle.

In Backward Dislocation there may be serious injury to the popliteal vessels and nerves

In Medial Dislocation the peroneal nerve is torn.

In Lateral Dislocation the tendons of semimembranosus, gracilis, semitendinosus, and sartorius slip back behind the medial condyle of the femur and get caught in the posterior intercondylar fossa

COMPLICATIONS.—

Rupture of either or both popliteal nerves.

Rupture of popliteal artery with gangrene

TREATMENT.—

Reduction under general anæsthesia. It may be necessary to flex knee in order to release tendons (semimembranosus, etc.) (*Fig* 108)

Splint.—Back splint with foot-piece, or Cramer's wire splint if there is much swelling. Aspiration of the joint if there is much effusion

Plaster Cast —For 2 to 3 months

SUBLUXATION OR INTERNAL DERANGEMENT

VARIETIES

Rupture of menisci.

Rupture of cruciate or collateral ligaments

Loose bodies.

Nipping of synovial fringes

Rupture of Menisci.—

CAUSES.—Violent rotation of the body standing on one leg, as, e.g., when a violent kick is missed Any sudden twist of the slightly bent knee.

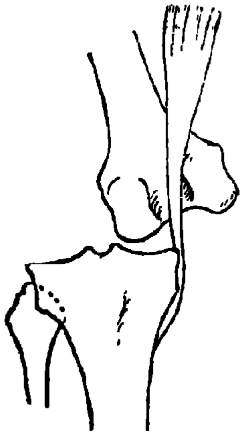

Fig. 108.—Dislocation of knee.

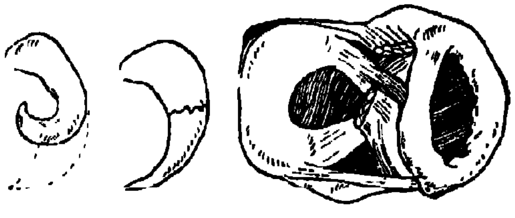

Fig. 109.—Three common varieties of rupture of the internal semilunar cartilage of the knee. *a*, Rupture of anterior coronary ligament. *b*, Transverse rupture. *c*, Longitudinal rupture; deep portion displaced towards centre of joint ('bucket-handle' rupture).

ANATOMY.—

Normal.—Both menisci are attached to the tibia by: (1) Their two extremities; (2) Short vertical ligaments—the coronary ligaments—to the edges of the tuberosities. The medial is semicircular; it is firmly attached to the capsule of the joint and to the medial collateral ligament. The lateral meniscus is nearly circular; the greater part of its edge is separated from the capsule by the tendon of the popliteus muscle. Hence the medial cartilage is more fixed, the lateral more mobile.

In Rupture.—The medial meniscus is affected twenty times more often than the lateral. The reasons for this are:—

1. The greater fixity of the medial cartilage.
2. The fact that normal slight rotation of the knee-joint takes place round the lateral condyle as a pivot, the medial tuberosity and cartilage moving, whilst the lateral are stationary.

The meniscus or its ligaments are torn in one of the following ways:—

1. One or both extremities from the tibia.
2. The edge from the capsule and medial collateral ligament.
3. The coronary ligament from the tibia (*Fig.* 109).
4. The cartilage divided into two pieces, either across or parallel to its long axis (*Fig.* 109)
5. The medial collateral ligament itself is often partly torn
6. A part of the cartilage may be torn right off to form a loose body.

The torn cartilage becomes displaced, so as to be locked between the joint surfaces or in the intercondyloid notch.

Synovial effusion with synovitis results.

Late Changes in the Joint.—The torn cartilage and ligaments become converted into fibrocartilaginous tags or loose bodies in the joint. Chronic synovitis is caused by repeated attacks of synovial effusion following a locked joint. The ligaments become relaxed and weakened. The synovial membrane becomes thickened and its free edges converted into fibrous fringes

SIGNS AND SYMPTOMS.—

Violent pain in the knee-joint, chiefly on the inner side over the lateral ligament.

The knee is locked in a semi-flexed position.

Synovial effusion with its usual signs appears within a few hours.

Later, and in the absence of proper treatment, recurrent attacks of synovitis often occur in any action of flexion and rotation of the knee

Tenderness is most marked on pressure over the tibial attachment of the medial collateral ligament.

TREATMENT.—

Reduction by flexion and extension of the joint.

Immobilization.—This may be done by a plaster cast, or a Thomas's walking calliper. Absolute fixation is maintained for three weeks. After this the splint is removed daily for active movements

Operation (for all recurrent cases).—If thorough immobilization has been neglected at first, it is useless later. Empty limb of blood by raising or using Esmarch's bandage; apply tourniquet above the knee. Incision. In clear cases, this should be oblique in front of the tuberosity. In doubtful cases a U-shaped incision is better, because then the posterior as well as the anterior part of the cartilage can be explored. Open the joint. Remove completely all injured or displaced parts of

Knee—Rupture of Menisci—Treatment, *continued*

the cartilage. Cut any tags of hypertrophied synovial membrane. If there is much oozing into the joint, it is wise to use a small drain of a few strands of silkworm gut, removed after twenty-four hours.

AFTER-TREATMENT —Active exercises of the quadriceps from the very first., Elastic bandage for the knee—walking from the eighth day.

RETENTIVE APPARATUS (when operation is refused or undesirable in chronic cases).—The mechanism embraces the joint, and whilst allowing flexion does not permit of any rotation.

Rupture of the Cruciate Ligaments.—Usually the result of great direct violence producing partial dislocation

RUPTURE OF THE ANTERIOR LIGAMENT —This ligament, which is attached to the lateral condyle of the femur and to the front of the tibia, is tense during extension of the knee When ruptured the tibia slips forward on the femur

RUPTURE OF THE POSTERIOR LIGAMENT —This ligament, which is attached to the medial condyle of the femur and to the back of the tibia, is tense during flexion of the knee Its rupture is accompanied by a backward displacement of the tibia on the femur.

TREATMENT —(1) Cage splint hinged at the joint. (2) Replacement of torn ligaments by fascia or tendons A strip of fascia lata is threaded through the lateral condyle and head of tibia for the anterior ligament, and the semitendinosus and gracilis tendons through the head of the tibia and medial condyle for the posterior ligament

Rupture of the Collateral Ligaments.—Usually the medial collateral. Causes instability and genu valgum or varum

TREATMENT.—By plaster cast for six weeks, followed by a walking iron jointed at the knee (*see* p. 209 and *Figs* 87, 92).

Loose Bodies.—(*See* p. 260)

Nipping of Synovial Fringes.—Usually follows chronic or recurrent synovitis. Pain and effusion follow some unusual movement, but there is no locking.

TREATMENT —Arthrotomy with removal of the hypertrophied synovia.

ANKLE

FOOT MAY BE DISLOCATED. (1) At the ankle-joint, with or without fracture; (2) At the astragalo-calcanean joint.

ASTRAGALUS MAY BE DISLOCATED from between the tibia and the os calcis.

Dislocation of Ankle-Joint.—

VARIETIES.—

OUTWARDS (the commonest) or INWARDS—always combined with Pott's fracture.

UPWARDS (the rarest)—generally with Dupuytren's fracture

BACKWARDS or FORWARDS—generally without fracture

BACKWARD DISLOCATION.—Both lateral ligaments or both malleoli ruptured. Heel projects backwards. Distance from malleoli to heel is increased. Distance from malleoli to toe is diminished.

FORWARD DISLOCATION.—**Structures ruptured as above. Heel is much shortened. Distance from malleoli to heel is diminished. Distance from malleoli to toes is increased**

UPWARD DISLOCATION.—Lateral and interosseous ligaments are torn. Malleoli are widely separated. Shortening of the leg.

TREATMENT.—Reduction and fixation as in Pott's fracture.
Massage and passive movements from third day.

Dislocation of the Astragalus alone.—

VARIETIES —

FORWARD.—Generally forwards and outwards May be forwards and inwards

BACKWARD

ROTATION on a sagittal axis, combined with forward or backward displacement

Generally incomplete—rarely complete.

SIGNS.—Prominence of the astragalus is felt on dorsum of the foot in forward dislocations—Above the heel in backward dislocations
Distance of malleoli from back of heel and from toes is not altered.
Distance of malleoli from sole may be lessened

TREATMENT —Manipulation under anæsthetic with knee flexed.
In complete cases or irreducible incomplete cases, open operation—Replace or remove the astragalus.

Subastragaloid (Astragalo-Calcanean) Dislocation.—

VARIETIES —

BACKWARDS (generally) —Backwards and inwards, or backwards and outwards.

FORWARDS (very rare)

SIGNS of the ordinary backward dislocations —
Head of astragalus is prominent on the dorsum Skin may be burst by the tension
Distance from malleoli to heel is increased. Distance from malleoli to toes is decreased
Foot is plantar-flexed, and inverted like equinovarus, or everted like equinovalgus

TREATMENT —Reduction under anæsthetic.
Open operation is necessary if reduction is impossible Free the tibialis anticus and posticus tendons from neck of the astragalus If this fails to reduce, excise the astragalus.

CHAPTER XXIII

DISEASES OF JOINTS

INFLAMMATION OF JOINTS

ACUTE SYNOVITIS

Causes.—Injury, e g., sprain or dislocation—Rheumatism and gout—Syphilis and gonorrhœa—Pyæmia

Anatomy.—Synovial membrane is hyperæmic. Plasma and cells exude (1) into the membrane, (2) into the joint Membrane is thickened and red. Endothelium may be shed.

Fluid in joint is increased—May be blood stained

Lymph is deposited on the articular surfaces

If the endothelial surface has been destroyed, as it generally is in septic cases, a plastic fibrocellular exudation takes its place This subsequently becomes organized into adhesions.

If the inflammation is severe, the ligaments become softened and relaxed

If the cause is simple and not septic, the exudation becomes absorbed, with few or no adhesions, except where cartilages or ligaments have been torn.

Signs.—

INFLAMMATION —Heat, redness, swelling, and tenderness

JOINT IS FIXED by muscular spasm. Position of ease assumed; generally slight flexion Active and passive movements are equally painful.

SYNOVIAL EFFUSION causes a fluctuating swelling, of characteristic shape. Muscles round joint atrophy (in severe and septic cases). Pain is not felt when the part is at rest Absence of tenderness on pressing the bony points.

Diagnosis.—

IN ARTHRITIS there are pain and swelling of bony ends. Pain is worse in active than passive movements. Grating shows destruction of cartilage

IN TUBERCULOUS DISEASE —Original injury absent or trivial—Slow onset—Muscular wasting marked—Inflammation slight or absent—Synovial swelling is pulpy, rather than fluid—May be signs of bone affection as in arthritis—Reaction to tuberculin

IN SPRAINS —Violent trauma—Immediate swelling—Marked ecchymosis.

IN HÆMOPHILIA.—History of bleeding and bruising—Rapid swelling after trivial injury—Comparatively painless—May be signs of creaking and grating.

Diagnosis of the Nature of Acute Synovitis.—(*See* SPECIAL FORMS OF ACUTE ARTHRITIS, p. 250.)

Treatment.—

1 Immobilization of joint, when effusion is increasing and inflammation is present.
2. Firm bandaging.
3 Aspiration if great tension exists
4. Ice or evaporating lotions in early stages Hot fomentations to relieve pain after twenty-four hours.
5. Massage to be used directly the swelling ceases to increase, and when the inflammation has gone.
6 Active movements as soon as they can be done without pain. Essential to maintain the tone of the muscles surrounding the joint by physiotherapeutic means
7 If adhesions exist so as to prevent movement, treat by weight extension until inflammation disappears. Move under anæsthetic, if no inflammation exists, through one full range of movement.

CHRONIC SYNOVITIS

Causes.—Previous acute attack. Causes of acute synovitis acting in a slight degree. Chronic irritation in joint (ruptured or displaced cartilage, loose cartilage, torn synovial fringes, foreign body) Want of tone in muscles and vessels; e g , after rising from long confinement to bed.

Anatomy.—Great distension of capsule. Stretching of ligaments. Distension of bursæ communicating with the joint. Thickened synovial membrane, the free edges of which are often produced into hypertrophied villous fringes. The cartilage is often rough and fibrillated

Signs.—Characteristic swelling Weakness in the joint. Easily fatigued. Little or no pain

Diagnosis of chronic synovitis to be made from other conditions producing HYDROPS ARTICULI, viz —

OSTEO-ARTHRITIS —Marked grating—Several joints generally affected —Lipping of the cartilage rim

CHARCOT'S DISEASE —As in the last, with signs of tabes or syringomyelia

TUBERCULOUS SYNOVITIS —Very rare as a hydrops—Absence of adequate cause—Marked muscular wasting—Tuberculous reaction.

SYPHILIS (secondary) and GONORRHŒA —The diagnosis rests on a discovery of the original disease

Treatment of Chronic Synovitis.—

1 Fix the joint, and apply firm pressure, with counter-irritation, e g , Scott's dressing
2. Try systematic massage, with hot-air baths
3 Aspiration, with pressure and rest.
4 Open the joint Remove foreign body or synovial fringes. Wash out with sterilized water

BAKER'S CYSTS

Definition.—Cysts originating from synovial pouches connected with joints. Most commonly seen behind the knee-joint.

Causes.—Chronic synovitis—Bursitis of bursæ connected with joints—Osteo-arthritis—Tuberculous synovitis.

Baker's Cysts, *continued.*

Anatomy.—Synovial sac buried in muscles and fasciæ. Connected to joint by a stalk which has an open communication with the joint, but this may become closed later (*see Fig.* 42, p. 162).

Signs.—Fluctuating swelling in the neighbourhood of a joint. The swelling can in some cases be reduced into the joint The joint itself usually shows signs of chronic synovitis

Treatment.—Excision if they cause pain.

ACUTE ARTHRITIS

All the joint structures are inflamed, i e, synovial membrane, ligaments cartilages, and bones

Causes.—

DIRECT INFECTION —(1) By wounds (2) By blood-borne infection — e g., streptococcal, staphylococcal

EXTENSION from infective osteomyelitis especially in syphilitic infants. Generally staphylococcal.

SPECIFIC FEVERS.—Measles and scarlet fever (streptococcal infection) —Enteric (*B typhosus*)—Pneumonia (*Pneumococcus*)—Gonorrhœa (*Gonococcus* or mixed infection)—Rheumatism (doubtful organism)—Pyæmia.

Symptoms.—Synovial distension Fixation of joint by muscular spasm Slight flexion of joint to give position of rest.

PAIN —Acute tenderness All movement is impossible Active movement is worse than passive Starting pains when patient falls asleep

SIGNS OF ACUTE INFLAMMATION —Heat, redness, and œdema

ABSCESS formation in the neighbourhood of joint. Abscesses burst and leave sinuses, leading to dead bone

SEVERE TOXIC FEVER

DISLOCATION occurs at a later date when the ligaments have given way.

Terminations.—

RECOVERY with a movable joint. only possible in cases treated very early

DISLOCATION following disorganization

ANKYLOSIS, fibrous or bony.

DEATH from septicæmia or pyæmia in acute stage, or secondary hæmorrhage.

Anatomy.—

SYNOVIAL MEMBRANE is acutely inflamed.

The exudation from the blood-vessels, instead of forming plastic lymph, is killed by bacterial toxins and forms pus

The endothelium and then the deeper layers are destroyed by this suppurative inflammation

The surface of the synovial membrane is eventually replaced by granulation tissue, which spreads rapidly as a 'pannus' on to the neighbouring cartilage, eroding and destroying it.

LIGAMENTS become softened by inflammatory infiltration. Those which are surrounded by synovial membrane (e.g., ligamentum teres in hip, or cruciate ligaments in knee) soon disappear, digested by the proteolytic action of leucocytes and bacteria. Others stretch or are ulcerated through.

CARTILAGE.—Colour changes to dull opaque yellow. It is eroded irrespective of pressure points. It is raised in flakes from the bone by inflammatory processes creeping from the edges or through perforations in its surface Ultimately the cartilage is almost entirely replaced by granulation tissue which springs from the bone beneath, and from the surrounding zone of granulations which have replaced the synovial membrane.

BONE.—The articular surfaces become bare of cartilage. Acute osteitis occurs. The bony tissue is absorbed by osteoclasts. The vessels and cellular elements increase, thus producing osteoporosis and caries. Areas of necrosis occur.

THE PERIOSTEUM is acute inflamed, but extensive necrosis does not occur, because the bony ends have an independent blood-supply.

In chronic cases which survive the acute stage, stalactiform masses of new bone are produced by the inflamed edge of the periosteum.

THE JOINT CAVITY is first filled with turbid serum; then with pus, and flakes of necrosed cartilage Is later lined by granulating surfaces which replace the synovial membrane and cartilage. The granulations coalesce and become organized into dense fibrous adhesions.

Treatment.—

1. IN EARLY, MILD, AND DOUBTFUL CASES.—

a. GENERAL —Good food and fluids, analgesics to relieve pain. Blood transfusions and chemotherapy in selected specific cases.

b LOCAL.—

Rest, and

Bier's Congestive Treatment —A rubber bandage is wound round the limb at some distance above the joint, sufficiently tight to obstruct venous and lymphatic return, without producing coldness or loss of pulse Kept on for twenty hours out of twenty-four. Limb is elevated during the interval It makes the whole limb fiery red and very swollen and œdematous. It relieves the pain at once. Produces resolution without suppuration in many cases. Aspiration of the joint relieves pain by reducing distension of joint and is also of diagnostic aid It may be combined with irrigation of the joint with lotio flavinæ or mercurochrome

2 IN CASES WITH PUS, BUT BEFORE THE ARTICULAR SURFACES HAVE BEEN DESTROYED —Free incisions are made into the joint and the cavity washed out Wounds are partially closed and a drain taken down to, but not into, the synovial cavity Active movement is encouraged, as this promotes drainage

3. IN CASES WHERE DESTRUCTION OF THE ARTICULAR SURFACE HAS PROBABLY OCCURRED.—Immobilization in best position for ankylosis. (Knee: 5° flexion. Hip: 30° flexion and slight abduction. Shoulder: 40° abduction. Elbow: At angle of 135°, and midway between supination and pronation. Wrist: Dorsiflexion. Foot: At right angles and slight inversion.) Weight extension prevents starting-pains.

Open the joint freely. Irrigate with sterile salt solution. Drain down to the joint but not into it.

Change the dressings frequently, with irrigation.

Acute Arthritis—Treatment, ***continued.***

4. AMPUTATION may be required: In toxæmia threatening life—In secondary hæmorrhage—In chronic suppurating sinuses.
5. EXCISION may be required: For ankylosis in a faulty position.

SPECIAL FORMS OF ACUTE ARTHRITIS

Rheumatic.—Generally a synovitis only. Complete recovery without ankylosis is the rule One joint after another is attacked.

Occasionally arthritis of one or more joints occurs Ankylosis without suppuration results.

Chronic synovitis is sometimes left. Synovial membrane becomes pulpy Adhesions produce fibrous ankylosis. Recovery may occur after a long time

Salicylates are most useful in acute cases.

Acute arthritis resisting salicylates should be opened and washed out.

Gouty.—Generally attacks big-toe joint first. Attack begins suddenly in the night Part is red, œdematous, and very tender. Seldom affects women Small rather than the large joints are affected. Leads to changes of osteo-arthritis

CAUSED by a deposit of biurate of soda in the cartilage, ligaments, and fibrous tissue of the joint.

ASSOCIATED with uratic tophi round the joints and in the ears

TREATMENT —By hot fomentations in the acute stage, and lithia, colchicum, and alkalis in the chronic

Pyæmic.—The joints become rapidly distended with pus. Marked absence of pain

TREATMENT —By opening, washing, and draining

Use of appropriate vaccine

Chemotherapy.

Enteric.—

1 Simple synovitis, probably toxic rather than microbic
2 True typhoid arthritis, producing destruction of ligaments and dislocation without suppuration. Tends to spontaneous recovery.

   Treatment. —By maintaining good position. Opening if the effusion becomes great
3. Mixed or pure pyogenic infections Symptoms and treatment as for acute arthritis in general

Pneumococcal.—Occurs as a complication of pneumonia, and less frequently of pneumococcal meningitis or peritonitis

IN ADULTS.—

JOINT INCIDENCE —Upper limb rather more frequently affected than the lower The knee much more often than any other joint. In two-thirds of the cases only one joint is affected; in about one-third, two or more suffer at once.

ANATOMY.—About 90 per cent suppurate, and the remainder present a sterile synovitis. Three conditions may thus be found:—

1. A serous synovitis—the rarest.
2. A purulent arthritis with thick flakes of lymph and clear fluid.
3. A purulent arthritis with thick or thin pus.

The pneumococci are found on the surface of the exudate. The cartilages are eroded and the ligaments destroyed in the acute cases, and if the patient lives, an extra-articular abscess forms. There is little tendency to bone destruction.

PROGNOSIS.—About 70 per cent of cases die from the toxæmia.

TREATMENT.—Incision and drainage. The functional results are good if the patient lives. Chemotherapy, using appropriate member of sulphonamide group.

IN CHILDREN.—

AETIOLOGY.—It may be a primary infection of the joints, or more often is secondary to otitis media.

ANATOMY.—Destructive joint changes are less marked than in the adult form

SYMPTOMS.—An irregular fever, with comparatively indolent swelling of the joint, are the chief features. Sleep is undisturbed, and the child may eat well.

SIGNS —The skin is pale, and there is a diffuse œdema round the joint extending, it may be, over the whole limb. There are tenderness and increased heat, but little redness Some cases may resolve without suppuration

TUBERCULOUS DISEASE OF THE JOINTS

Aetiology.—

PREDISPOSING CAUSES —Hereditary disposition (of slight importance). Injury to the joint by trauma or other forms of synovitis. Any condition of general debility

EXCITING CAUSE.—The entrance of the tubercle bacillus Sometimes this evidently comes from some other primary focus, but more often not. Usually the bovine type from infected milk.

AGE.—Patients of any age may be affected. Children and growing adults are the most susceptible, because the young cells of the growing parts of the bone form suitable tissue for the development of the disease. Old people are rather more susceptible than middle-aged, because of their lowered vitality.

POOR PEOPLE are specially liable, because of their bad food, defective hygiene, and the neglect of trivial injuries

Pathology.—The disease primarily affects either the synovial membrane or the ends of the bone The cartilages and ligaments are affected usually secondarily

SYNOVIAL CHANGES —

INFILTRATION AND DEGENERATION.—Pulpy swelling caused by œdematous thickening of the membrane, which is studded by early tubercles. Caseation of some of the tuberculous foci, which break into the joint or surrounding structures. Suppuration (in the tuberculous sense) This occurs only in some cases; in those treated early the disease never goes beyond the pulpy thickening or caseation. When it does occur, a condition like that of a chronic abscess is set up, the synovial membrane being replaced by a fibrous layer lined by granulations exuding tuberculous pus.

REGENERATION AND REPAIR.—The caseous foci become calcified or encapsuled by fibrous tissue. The granulations are converted into fibrous tissue Opposed granulating surfaces grow together to form fibrous adhesions

Tuberculous Disease of Joints—Pathology, ***continued***

- Extension always takes place to some of the other tissues.
  - Ligaments are attacked, softened, and destroyed very early, because the synovial membrane is so closely related to them.
  - Cartilages are invaded by a 'pannus' of tuberculous granulation tissue creeping over them and the disease spreading under their edges.
  - Bones are attacked first under the edges of the articular cartilages, and when the latter have been destroyed, directly from the joint disease.
  - Bursæ which are connected with the synovial cavity are infected, and in chronic cases may form 'Baker's cysts'.
  - The Cavity of the Joint is first distended with synovial fluid, but a true hydrops articuli of tuberculous origin is rare.
  - The Pulpy Membrane may be greatly hypertrophied and form fringes which fill up the folds of the joint, and may cause loose bodies from a separation and fibrous thickening of the free extremities.

BONE CHANGES.—

- Point of Origin.—(1) Beneath the articular cartilage; (2) In the epiphysis next to the growing cartilage; (3) In the diaphysis next to the growing cartilage (this is rare, except in the neck of the femur).
- Nature —Tuberculous invasion of the cancellous tissue Rarefying osteitis or caries of the bone Necrosis of small islands of bone between or in carious foci. Osteosclerosis or reparative osteitis round the outer margin of disease tends to limit its spread. The usual tuberculous events of caseation or suppuration accompany its development.
- Spread —Under the edges of the articular cartilage into the joint Through the articular cartilage into the joint Through the epiphysial cartilage into the shaft Through the compact bone of the neck into the tissues outside the joint

CHANGES IN THE CARTILAGES (*see Fig* 111, p 265).—These are always secondary to synovial or osseous disease

- Primary Bone Disease beginning under the articular cartilage causes their earliest and most complete destruction
- Attacked from their Deep Surface, by the tuberculous granulations in the bone, the latter fungate through them, or more rarely separate off flakes as loose bodies in the joints
- Attacked from their Superficial Surface, as in synovial disease, they become eroded by the overlying tuberculous granulations from their edges towards their centres, and finally quite replaced by this granulating surface

CHANGES IN THE LIGAMENTS —The ligaments also are only secondarily affected, and especially in synovial disease They are attacked and replaced by tuberculous tissue They then quickly become soft and disappear.

- Ligaments surrounded by synovial membrane will suffer early and completely, because they are attacked on all sides, e.g., the ligamentum teres of the hip, or the cruciate ligaments of the knee
- This destruction of the ligaments allows pathological dislocation to take place by the action of gravity or of muscular tension.

CHANGES IN THE PERIOSTEUM.—This is not as a rule much affected, because it ends at the junction of epiphysis and diaphysis outside the area of disease. It may become inflamed and form new spicules of bone like stalactites round the diseased area. This is more likely to happen if secondary pyogenic infection has taken place.

Change in the Joint as a Whole.—These will depend upon: (1) The structures attacked first; (2) The severity of the process, (3) The efficiency of treatment, (4) The anatomical structure of the joint; (5) The occurrence of pyogenic infection.

There are four chief conditions found in the active stage of tuberculous joint disease:—

1. DISTENSION OF THE CAPSULE by pulpy synovial membrane and fluid, the ligaments and cartilages being intact
2. DISLOCATION from destruction of ligaments, or more rarely of the articular surfaces of the bones, aided by neglect of treatment.
3. DESTRUCTION OF THE BONE ENDS.—The cartilages have disappeared, the diseased bones may be carious or necrotic, forming sequestra in the joint, or they may give rise to TUBERCULOUS ABSCESSES which track outside the joint limits.
4. PYOGENIC INFECTION through opened or burst abscesses

Ultimate Effect upon the Joint may be one of the following:—

1. RECOVERY.—This is possible only when the original disease has been limited to the bone, or when a synovial affection has been arrested or excised in a very early stage
2. FIXATION BY ADHESIONS—This results from the destruction of synovial membrane and cartilage, and their being replaced by granulations which have formed fibrous adhesions
3. FIBROUS ANKYLOSIS.—This results from a more or less complete destruction of the articular cartilages, followed by a union of opposed granulating surfaces of diseases bone.
   Bony ankylosis only occurs if secondary infection takes place (*see Fig.* 112, p. 265).
4. DISLOCATION.—The diseased tissues recover, but the dislocation remains, and usually the parts become fixed in a mass of fibrous tissue.

Symptoms.—The clinical history can conveniently be divided into three stages, preceded by a latent period, but of course all these stages cannot be recognized in every case

LATENT PERIOD—A history is given of some slight injury, since which there has been apparently complete or partial recovery, with a disinclination to use the joint. Change in temperament of small children—fretfulness and peevishness

EARLY, 'FIRST', STAGE, OR STAGE OF INVASION of the disease. Here the disease is limited to either the bone or synovial membrane in which it started, and has not spread to the other structures.

a IN SYNOVIAL DISEASE.—A distension of the joint cavity and formation of a 'white swelling'. The joint is held in a position which allows of the greatest distension of the capsule, e g., flexion The muscles in the neighbourhood are wasted. All movement is painful, passive as well as active Very rarely a fluctuating hydrops articuli may exist.

b. IN BONE DISEASE.—The bone may be swollen; it is tender on pressure. Pushing or jarring the joint surfaces together is painful. There is no fixation in any special position, and passive movements are relatively painless. Active movements or placing any weight on the limb are

Tuberculous Disease of Joints—Symptoms, ***continued.***

painful, because all active movements 'crowd together,' the joint surfaces. A radiograph will show a light area in the bone end.

SECOND STAGE, OR STAGE OF FIXATION.—Here the ligaments and cartilages have been infected from the primary focus, and therefore all the joint tissues are affected. All voluntary movement is abolished by pain. The muscles hold the joint by a tonic contraction, in which the most powerful groups gradually overcome the others, thus producing a fixed deformity Starting pains occur at night, especially when the cartilage is eroded, by the relaxation of this tonic contraction, with consequent jarring of the joint surfaces.

THIRD STAGE, OR STAGE OF DESTRUCTION.—The ligaments have given way, the cartilages are lost, and the bones are carious or necrosed, whilst fixation, wasting, deformity, and pain continue. Further destruction of the joint is manifested by· (1) Actual shortening of the bones, (2) Abscess formation; (3) Dislocation

If the abscesses open upon the surface and become septic, amyloid disease may ensue.

CONSTITUTIONAL SYMPTOMS —These may be entirely absent. Loss of appetite, general malaise, with loss of flesh, are often seen in acute or extensive cases. A hectic temperature is rarely seen unless a pyogenic infection has been added. But it sometimes occurs in children, and then indicates extensive bone disease

Complications and Sequelæ.—Phthisis—Tuberculous peritonitis—Meningitis—General miliary tuberculosis (all rare except the first)—Septicæmia or amyloid disease when an abscess has become infected with pyogenic organisms.

Prognosis.—This is on the whole good Bad · (1) When treatment cannot be efficiently carried out, (2) In children under three and patients over fifty; (3) In the presence of any visceral complication; (4) When septic complications have occurred

Treatment.—

1 CONSTITUTIONAL (*see* TUBERCULOSIS, p 57). Sanatorium treatment, fresh air, good food, heliotherapy, adequate rest

2. REST.—The diseased joint must be protected from movement and from pressure, which aggravates the disease and its consequences. Bed for a short time and splints for a long time. Complete immobilization must be maintained for some time after all pain and tenderness have disappeared Three to eighteen months is usual.

3. CORRECTION OF DEFORMITY.—This is to be carried out as early in the disease as possible. It must be done gradually, e g., by weight extension Traction sufficient to abolish pain, in the line of the deformity. It aims at placing the joint in the most useful position if ankylosis results (*see* p. 249).

4. WEIGHT (OR ELASTIC) EXTENSION.—This tends to correct early deformities; to counteract the tonic contraction of the muscles which press the joint surfaces together: to prevent dislocation in the later stages. It is specially useful in diseases of the spine, hip, and knee It is followed by immobilization either by plaster-of-Paris or in a special frame.

5. LOCAL TREATMENT.—To be carried out in conjunction with the above:—
   a. **Passive Hyperæmia.** Produced by an elastic band above the joint, applied for one hour daily.
   b. **Aspirating Abscesses** under careful aseptic precautions. In most cases, however, the presence of an abscess is an indication for radical operative treatment.

5. OPERATIVE TREATMENT.—Required: (i) When conservative treatment fails. (ii) To shorten the time of treatment—e.g., adult patients with tuberculosis of the wrist, in which it is better to get a sound bony ankylosis than a fibrous type from conservative treatment. (iii) To improve position. (iv) To regain mobility, e.g., in the elbow

   Operative measures are:—
   a. **Partial Excision** or arthrectomy is an operation of very doubtful utility. If it succeeds in curing, then the disease would probably have been curable without any operation. It usually results in recurrence or ankylosis, with subsequent deformity, requiring a complete excision. It consists in removing diseased synovial membrane with a minimum of ligaments and cartilages, so as to preserve a movable joint.

   It is indicated only in young children (under ten) where there is no bone disease, and where careful non-operative treatment is impracticable.

   b. **Arthrodesis.**—Either intra- or extra-articular, or a combination of both. Extra-articular arthrodesis is of great value in the hip and spine. It is indicated in cases where there is much pain

   c. **Complete Excision**, i.e., removal of the bony ends, all cartilages, and most of the ligaments, as well as the synovial membrane.

   It is indicated: (i) In cases in which general treatment is impossible or has failed. (ii) When there is definite evidence of bone disease which has involved the joint (iii) When abscesses have formed, especially when these arise from bone disease (iv) To cure ankylosis in joints, e.g., the shoulder or elbow (v) To correct faulty ankylosis, e.g., in the knee.

   d. **Amputation.**—This is rarely called for

   It is indicated: In condition of very bad general health In elderly patients, especially when the ankle-joint is diseased. When incurable sepsis, with possibly lardaceous disease, has supervened. When excision has failed. When so much bone is diseased that excision would leave a useless limb When two joints are affected in the same limb

SYPHILIS OF JOINTS

Joints are very rarely affected by syphilis, though there are many ways in which syphilis in its various stages may affect them

Classification.—

SECONDARY STAGE.—Arthralgia. Synovitis—intermittent and painful, or chronic and painless.

TERTIARY STAGE.—Gummatous synovitis. Chondro-arthritis Virchow's ulcerating joints. Tabetic—Charcot's sclerosing joints.

INHERITED DISEASE. — Suppurative arthritis—in early infancy. Hydrarthrosis. Symmetrical serous synovitis (Clutton's). Gummatous synovitis. Chondro-arthritis (von Gies' joints). Charcot's joint (juvenile tabes).

Syphilis of Joints—Classification, *continued.*

1. CHRONIC SYNOVITIS.—Usually symmetrical, occurs in the late secondary stage; most often in the knee-joints. These become distended by effusion, with little or no pain
2. CHRONIC (GUMMATOUS) ARTHRITIS.—Gummata may be:—
   - *a.* EXTRA-ARTICULAR.—A chronic swelling outside a joint, which softens and bursts externally
   - *b.* INTRA-ARTICULAR —The synovial membrane is the seat of a local or diffuse gummatous change. A condition is produced very like tuberculous joints It differs from the latter by being almost painless, and by producing very little destruction of the ligaments and cartilages.
3. OSTEOCHONDRO-ARTHRITIS —May occur in the late acquired or in the congenital disease It resembles osteo-arthritis The cartilages become fibrillated and eroded The synovial membrane is thick and pulpy; the bony ends are thickened by periostitis It differs from osteo-arthritis by causing little pain, and occurring usually in much younger subjects The cartilages are 'punched-out' rather than worn away, and there is no lipping at their edges

Treatment.—Mercury and iodides by mouth. Firm pressure over mercury ointment applied to the joint

Excision is seldom required only in those cases where actual bony destruction has occurred.

ARTHRITIS DEFORMANS

A variety of joint lesions is included under this heading. Aetiology: obscure. Predisposing factors, trauma, infection, old age, toxic agents. Although no well-defined groups, classified as: (1) Acute polyarticular; (2) Chronic polyarticular, (3) Chronic monarticular

Aetiology.—The chief groups of causative conditions are:—

DEPRESSION OF THE VITALITY —Old age Constant cold and damp.

TOXIC.—Some pre-existing infectious disease, e g , rheumatism, scarlet fever, tonsillitis, influenza, or dental sepsis Auto-intoxication by some morbid digestive products in, e g , catarrh or ulceration of the intestines Infection by a short bacillus (Bannatyne) is demonstrable in many cases.

TRAUMATISM often acts as the exciting cause when the above-mentioned predisposing causes may have been present A single severe injury may rupture the cartilage and produce a monarticular osteo-arthritis at once Long-continued overstrain may bring about the condition slowly (e.g., in certain joints in labouring men)

Types of Lesion.—

1. ACUTE AND SUBACUTE POLYARTICULAR (e.g., Rheumatoid Arthritis).—Either toxic or infective in origin Young females, and small joints are affected, usually hands and feet. Characteristic lesion is thickening of the synovial membrane and the extrasynovial tissues, causing fibrosis with contractions and deformities.

   PATHOLOGY.—Early stages: Inflammatory changes in synovia—hypertrophy of the villi—small effusion into the joint Resembles low-grade inflammation. Later the synovial membrane and peri-articular structures become fibrotic. Cartilages not affected at first, but later a 'pannus' of granulation tissue grows over the articular cartilage, which is eroded and softened. Replaced by fibrous tissue. Underlying bone is rarefied. No osteophytic bone formation.

Signs and Symptoms.—Insidious onset, small joints of hands and feet. Joints swollen, shiny, and spindle-shaped. Pyrexia to 103°, patient is ill, general health is impaired, sleep disturbed by pain. Exacerbations occur. X-ray in early stages shows no bony changes Later, bones decalcified, translucent area near the joint surface. Diminution of joint space. (Fingers undergo characteristic ulnar deviation.)

Prognosis.—Poor, tends to progress in spite of treatment.

Treatment.—Eliminate any toxic focus, as in teeth, sinuses, tonsils, prostate, Fallopian tubes. Vaccines of little use. Attend to general bodily health. Splintage of joints to correct and prevent deformities. Spa treatment when acute stage has subsided.

Protein shock therapy—intravenous T A B. of value.

Local Treatment.—Radiant heat, diathermy, wax baths, etc., are of value.

Still's Disease.—Occurs in children—resembles above—associated splenomegaly.

2. CHRONIC POLYARTICULAR—Middle-aged females, small joints. Changes are degenerative, with atrophy of bone ends and fibrosis of the ligaments Localized patches of periostitis occur in the bone ends.

Treatment.—As above.

3 CHRONIC MONARTICULAR.—Trauma is the main factor. It is not an infective, but a degenerative change. Associated toxic conditions make it worse. Injury of a single nature, or results from minor repeated traumata from a displacing cartilage or a loose body, e.g., knee-joint. Excessive use, alteration in the alinement of the joint, and frequent exposure to damp and cold are predisposing factors. Affects mainly large joints, as knee and hip.

Pathology.—Affects soft tissues first, synovial membrane thickened, villiform processes develop which may be nipped in the joint on movement—lipoma arborescens Cartilage undergoes a fibrous metaplasia, fibrillation occurs, cartilage softens and from pressure is worn away, exposing bare bone which becomes dense and smooth—eburnation. Cartilage at the edge of the articular surfaces proliferates and is raised into ridges which become ossified to form osteophytes which may become detached. Bursæ in communication with the joint enlarge, e g., Baker's cyst in the knee. Recurrent effusions occur in the joint

Clinical Features.—Patient past middle age, stiffness and pain in the joint, stiffness is increased by rest. Later creaking or locking of the joint. Muscular wasting occurs. Symptoms undergo exacerbations.

X-ray—Early stages little to see, later diminution of the joint space, and later still osteophytic outgrowths occur. Loose bodies may be seen in joint.

Treatment.—Eradication of any septic foci. Analgesics, salicylates, etc. Protection of joint from trauma and cold. Rest only if painful joint, movements should be resumed as soon as can be tolerated. Radiant heat and diathermy relieve the pain and allow more movement. Manipulation of the joint through one complete range of movement under anæsthesia, followed by physiotherapy, affords relief.

Arthritis Deformans—Chronic Monarticular, *continued.*

In later stages operative treatment necessary; ankylosis of the joint giving a painless stiff joint is better than a painful partially mobile joint. (*See* special joints)

PROGNOSIS.—Is almost hopeless. The disease may be arrested, but hardly ever cured. It has little or no tendency to shorten life.

CHARCOT'S DISEASE

(*Neuropathic Arthritis*)

Definition.—An advanced condition of osteo-arthritis resulting from a definite nerve lesion, usually in the spinal cord

Causes.—Tabes dorsalis (in the great majority of cases)—Syringomyelia—Spina bifida—Hemiplegia, paraplegia, or neuritis. Some actual injury, or the ordinary mechanical strain on the joint, may act as the proximate cause.

Anatomy.—All changes of osteo-arthritis occur in an exaggerated form.

a. THE ATROPHIC FORM shows marked loss of cartilage and bone, and no osteophytes.

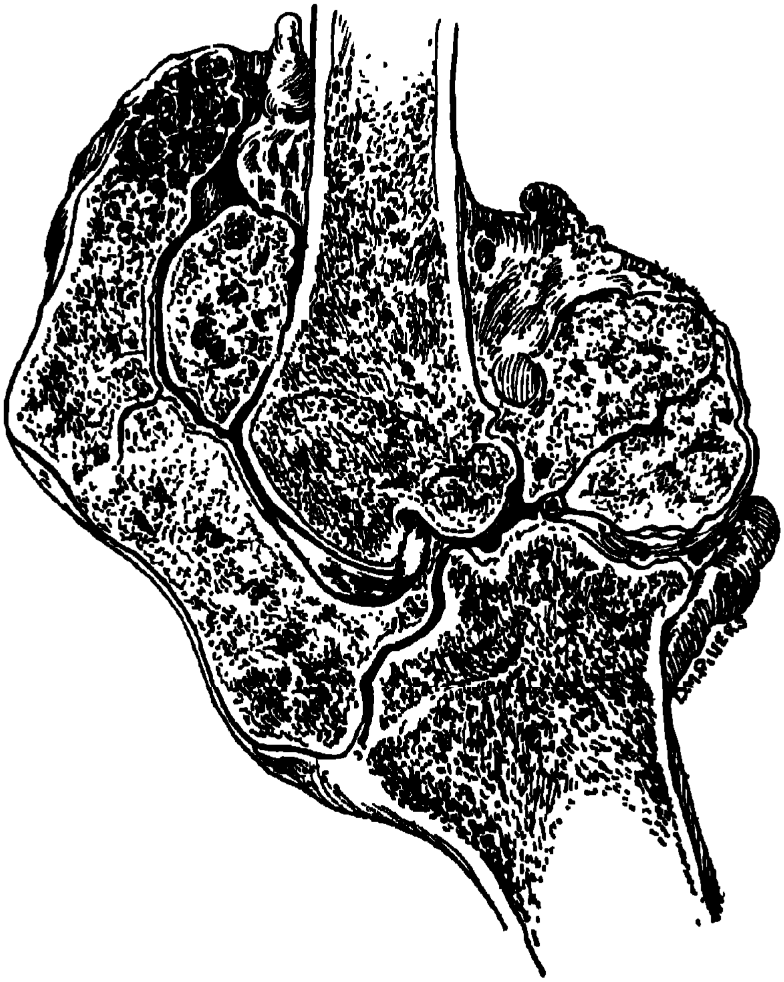

Fig. 110.—Charcot knee-joint.

b THE HYPERTROPHIC FORM shows exuberant overgrowth of cartilage and bone at the margin of the joint. Great increase of synovial fluid, and ultimate disorganization of the joint, are marked features. There is also a great tendency to the formation of osteophytes, which arise in and around the joint by an ossification of periosteum, synovial fringes, and muscular insertions.

Symptoms.—

NERVE SYMPTOMS.—Lightning pains. loss of knee-jerks. pupil changes. some anæsthesia or paræsthesia

JOINTS AFFECTED—The knee (*Fig.* 110), hip, shoulder, and ankle are affected in the order of frequency given. Very rarely affects more than one joint.

JOINT SIGNS.—

Very rapid and painless onset usually occurs

A great synovial distension is the first feature

All the signs of osteo-arthritis, viz., enlargement of the bone ends, lipping of articular margins, coarse grating, quickly follow.

Marked absorption of bone, with consequent shortening or deformity.

Massive heaping up of new bone as an outgrowth round the articular margins, in the hypertrophic varieties.

Disorganization or dislocation of the joint from a yielding of the ligaments and destruction of the joint surfaces

Absence of all pain or tenderness is a conspicuous and characteristic feature

Treatment.—Protection from injury Aspiration of excessive effusion. Some kind of mechanical support to prevent disorganization of the joint.

HÆMOPHILIA

Hæmorrhage into the joints causes two varieties of lesion:—

1 IN THE EARLY STAGES. Chronic synovitis
2 IN THE LATE STAGES as the result of repeated hæmorrhages. Osteo-arthritis.

Incidence of the Affection.—The joints are affected in the same order of frequency as in the case of ordinary traumatism: the knees much more frequently, then the ankles, wrists, elbows, shoulders, and hips A blow or strain which in a normal person produces a sprain, in a hæmophilic causes a hæmarthros Subsequently recurrent synovitis may occur without hæmarthros

Pathology.—The synovial cavity is distended with blood. The blood remains unabsorbed for a very long time Some deposit of fibrin takes place on all the articular surfaces. The chronic irritation thus set up produces: in the cartilages—fibrillation, absorption, and nodular thickening at margins, in the synovial membrane—thickening, with the formation of fibrous fringes; in the joint cavity—a tendency to chronic and recurrent synovitis; in the ligaments—relaxation.

Symptoms.—Acute synovial distension, with heat and pain, are the evidences of the first onset of hæmarthros. This differs from an ordinary synovitis only in its degree in proportion to the extent of injury, and its slowness to subside.

Hæmophilia—Symptoms, ***continued.***

Chronic and recurrent synovitis, especially in the knee-joints, usually follows repeated attacks of hæmarthros.

The usual signs of osteo-arthritis of the chronic variety finally supervene. Well-marked grating is felt, and the edges of the articular surfaces are lipped.

Treatment.—Of the hæmarthros—complete and prolonged rest, with firm pressure and cold applications. Of the recurrent synovitis—an elastic bandage constantly applied to the joint. Avoid any surgical interference, e.g., tapping.

LOOSE BODIES IN JOINTS

Varieties.—

FIBRINOUS, or 'melon-seed' bodies, formed in conditions of chronic synovitis, sometimes tuberculous

HÆMORRHAGIC —By the organization of blood-clot.

CARTILAGINOUS.—By a chipping off of parts of the articular cartilages or of ecchondroses found in osteo-arthritis. They form mulberry-like masses, from the size of a pea to that of a walnut.

BONE.—(*See* OSTEOCHONDRITIS DISSECANS *below.*)

SYNOVIAL —The hypertrophied fringes of chronic synovitis may become detached and form fibrous or cartilaginous loose bodies.

OSTEOCHONDRITIS DISSECANS.—Most common in the knee. A portion of the bone covered by cartilage, about the size of a sixpence, undergoes 'quiet necrosis', probably as the result of a blow followed by vascular thrombosis The whole fragment separates and lies as a loose body in the joint Both the loose body and the pit from which it fell out can be seen by X rays.

Number.—Usually single, may be multiple, and rarely number several hundreds.

Distribution.—Common in the knee, occasional in the elbow, and very rare in any other joint

Symptoms.—Sudden attack of acute pain, with momentary locking. Often followed by some synovitis. The loose body can often be detected, generally on the inner side of the patella. Chronic synovitis with relaxation of the ligaments supervenes if it is allowed to remain. Monarticular arthritis may develop

Treatment.—Removal through an open incision, which is much easier if the body can first be transfixed.

Diagnosis.—From displaced semilunar cartilage and nipped synovial fringes (*see* pp. 242–4).

ANKYLOSIS OF JOINTS

False Ankylosis.—The fixation or stiffness of a joint produced by extra-articular lesions. These are:—

1. CONTRACTION OF THE SKIN and soft parts, e.g., after burns.
2. CONTRACTION OF TENDONS in congenital, paralytic, or traumatic conditions, or after long-continued displacement.

3. THE MATTING TOGETHER of the soft parts by an injury or disease.
4. THE OSSIFICATION OF MUSCLES—myositis ossificans.
5. OUTGROWTHS of the bone, e.g., osteomata, chondromata, or sarcomata.

True Ankylosis.—The fixation or stiffness of a joint produced by intra-articular lesions.

1. FIBROUS ANKYLOSIS.—Formed by fibrous adhesions. The fixation is only partial, but attempts at movement produce pain. The causes are:—
   - *a.* SYNOVITIS which has resulted in synovial adhesions, especially tubercle, gonorrhœa, rheumatism.
   - *b.* TRAUMATISM.—This generally produces an extra- as well as an intra-articular ankylosis.
   - *c.* ARTHRITIS.—The cartilages have been to some extent replaced by granulation tissue, which has organized into fibrous adhesions. Especially met with in tuberculous conditions
   - *d.* NERVE LESIONS, e g., syringomyelia, or the division of, or neuritis of, a peripheral nerve.
   - *e* LONG-CONTINUED ABNORMAL PRESSURE of contiguous bones, e.g., in scoliosis or talipes.
2. BONY ANKYLOSIS —Formed by the complete destruction of the articular cartilages and the union of adjacent bony surfaces
   - *a.* ACUTE SUPPURATIVE ARTHRITIS, either septic or gonorrhœal.
   - *b.* CHRONIC TUBERCULOUS ARTHRITIS, especially if secondarily infected.

Diagnosis of the nature of ankylosis —

IN FALSE OR EXTRA ARTICULAR ANKYLOSIS there are signs of extra-articular injury or disease, e.g., swelling in the muscles, tendons, or bones. Movement is generally free and painless, but very limited in extent

IN FIBROUS ANKYLOSIS —history of some traumatic, inflammatory, or infective disease. Some movement is possible, but is very painful and accompanied by creaking. There are often swelling and thickening of the synovial tissues.

IN BONY ANKYLOSIS there is absolute rigidity of the joint, and attempts at movement cause no pain.

Treatment.—

MASSAGE, PASSIVE AND ACTIVE MOVEMENTS, WITH HOT-AIR BATHS.—All evidence of active disease, e.g , increasing swelling, heat, redness, or tenderness, must be absent. Should always be tried in the early stages, after the subsidence of active exudation or disease.

FORCIBLE MOVEMENT UNDER AN ANÆSTHETIC, followed by massage and passive movements, etc. In the same class of case as the above, after other measures have failed. When passive movements are very painful. It is of the utmost importance that all active inflammatory disease should be at an end. It is dangerous in all recent tuberculous cases.

GRADUAL CORRECTION OF THE DEFORMITY by e g., weight extension. Specially useful in tuberculous disease of hip and knee.

OPERATION OF ARTHROPLASTY.—If all active disease is at an end, the joint may be mobilized by separating the adherent articular surfaces,

Ankylosis of Joints—Treatment, ***continued.***

removing some bone from each, and then sewing a piece of fatty fascia between these surfaces. This will answer best in the hip, where the socket is deep and movements are possible at an early stage.

EXCISION OF THE JOINT TO PRODUCE MOBILITY.—Most often used in ankylosis of the elbow and shoulder. The bone is freely removed, and movements are begun a week later.

EXCISION OR OSTEOTOMY, WITH FIXATION.—Used most often in the hip- and knee-joints. For conditions of bony ankylosis in a bad position, or fibrous ankylosis which is painful or weak.

DISEASES OF SPECIAL JOINTS

SHOULDER-JOINT

Incidence of Disease.—Inflammatory diseases and tuberculosis rare. Osteo-arthritis very common Ankylosis (usually extra-articular) very common after traumatism, brachial neuritis, and osteo-arthritis. This is due partly to the facility with which the movements of the scapula replace those of the shoulder, and so prevent active and passive movements.

Synovitis produces a fullness of the deltoid and under the axilla ; best marked along the line of the bicipital groove

Must be distinguished from subdeltoid bursitis, which does not project into axilla, and causes little limitation of movements

Supraspinatus Lesions.—Pain and limitation of movement. especially of first 5°–10° of abduction Follows a trivial injury or overstrain Tendon of the supraspinatus has been torn May become calcified, when it shows in the X-ray

TREATMENT.—Open operation and repair of tendon or removal of nodule.

Tuberculosis usually begins in the head of the humerus, and may lead to purely extra-articular disease May take the form of a chronic caries sicca. Abscesses point along the biceps tendon or under the posterior margin of the deltoid.

TREATMENT —Is expectant in most cases.

OPERATION.—Is indicated when abscesses have formed, or where the radiograph shows extra-articular disease. If excision is done in children (very rare), as little of the epiphysis should be removed as possible.

Ankylosis.—This is usually fibrous, resulting from trauma.

TREATMENT —

Excision gives a movable joint but a weak arm

Arthrodesis is operation of choice if position of arm is bad (fixed to the side) and if the scapula is freely movable Articular surfaces are removed. and the acromion is broken downwards and nailed to head of humerus Arm is fixed with hand opposite face in 90° of abduction slightly anterior to the coronal plane

Flail Shoulder.—Due to certain paralyses—generally infantile.

TREATMENT.—By arthrodesis as above, provided the muscles which move the scapula and the hand are functioning.

ELBOW-JOINT

Synovitis.—Produces a fluctuating swelling behind on either side of the triceps tendon, together with an obscuration of the head of the radius. The joint is held in a flexed and pronated position.

Tuberculosis.—Very common. The joint cavity is always involved because of the smallness of the epiphyses. Early swelling and position as in synovitis. All three bone ends become diseased.

TREATMENT.—Is usually conservative, especially in children.

Opening and scraping carious foci, with the removal of the synovial membrane, is done in adults and in children in whom abscesses have formed. *A formal excision* is reserved for cases where painful or useless ankylosis has occurred

Ankylosis.—This is common after complicated fractures and after suppurative or tuberculous disease

If the position is one of flexion, it is a matter for the patient's opinion whether anything should be done; if the position is straight, operation is necessary.

Excision, with free bone removal, gives good results

WRIST-JOINT

Incidence of Disease.—Gonorrhœa, osteo-arthritis, and tubercle are common, but all other diseases are rare

Synovial Disease and distension produce a swelling, chiefly at the back of the wrist, but an extension to the neighbouring tendon sheaths soon obscures it. The joint is held flexed.

Tuberculous Disease is very chronic and difficult to cure owing to the number of bones and joints involved Pulpy swelling is well marked behind and at the sides of the joint, and extends downwards over the metacarpals. Pain is constant and severe in the later stages.

TREATMENT.—Is conservative and palliative to the utmost degree.

Only actual abscesses justify operation. Limited erasions should repeatedly be tried before an extensive excision In the worst cases amputation is required

ANKYLOSIS OF THE WRIST should always be ensured in a position of dorsiflexion.

SACRO-ILIAC JOINT

Incidence of Disease.—Pyæmic and tuberculous affections are practically the only diseases which affect this joint, and the former presents no special features.

Tuberculous Disease usually affects young adults, and is very rare in children.

SIGNS.—

PUFFY SWELLING over the joint behind.

APPARENT LENGTHENING OF THE LEG.—The anterior superior iliac spine is lowered on the affected side.

ABSCESS forms, and points over joint behind, in the gluteal region, through the sacro-sciatic foramen, behind the trochanter, in the pelvis, into the rectum, vagina, or perineum.

PRESSURE over the joint, pressing the two ilia together, or forcing them apart, is very painful.

Tuberculous Disease of Sacro-Iliac Joint, *continued.*

SYMPTOMS —A general sense of weakness on standing, walking, or straining A sensation of the pelvis breaking in two. Pain is often referred down the sciatic nerve. The signs and symptoms may be quite latent if only the peripheral parts of the joint are affected.

TREATMENT —Absolute rest until all active disease is ended
Opening over the joint posteriorly when abscesses have formed together with evacuation of the abscesses themselves. Bone grafting, so as to fix the joint. A strut or plaque of bone is fixed over the back of the sacrum and ilium after these have been bared
A short hip spica is applied to immobilize the joint.

HIP-JOINT

Simple Synovitis.—This is a rare condition. It gives the same signs as the early stage of tuberculous disease, and under rest with extension quickly passes off. Any tendency to chronicity or recurrence should be regarded as suspicious of tubercle.

'Snapping' Hip.—

INCIDENCE —An affection of young patients (fifteen to twenty-five), usually women.

SYMPTOMS —On flexing the hip and then putting weight on the leg (as on going upstairs), an audible, visible, and palpable click occurs, the leg is everted. and the great trochanter becomes more prominent. It may be painful and sometimes makes the patient fall. The sign cannot be elicited when the patient is lying down (i.e., when the muscles are relaxed) It may be bilateral.

PATHOLOGY.—It is caused by the band of fascia lata into which the gluteus maximus is partly attached slipping over the great trochanter when the hip is flexed and an effort is made to extend it (gluteus action). A bursa sometimes forms over the trochanter.

TREATMENT —A lateral vertical incision about 6 in. long is made over the great trochanter A vertical incision is made through the fascia lata in the line of the trochanter, dividing the part of the fascia which gives attachment to the tensor fasciæ femoris from that into which the gluteus is inserted. The posterior band of fascia is divided transversely for about 1 in., and the angles of the fascia sewn back behind the femur. If a bursa lies over the trochanter it should be excised.

Acute Septic Arthritis.—Usually pyæmic, or may follow typhoid fever or one of the exanthems.
Great distension of the joint cavity. Dislocation backwards is the common tendency.
TREATMENT.—By weight extension and early evacuation of the abscess.

Subacute or Chronic Infective Arthritis.—This results from gonorrhœa or some other infection. It ends usually in ankylosis.
TREATMENT.—Weight extension to produce a straight leg (*see* ANKYLOSIS).

Tuberculous Disease of the Hip.—

AGE.—Five to fifteen is the commonest age to begin.

PATHOLOGY.—Disease may commence:—

1. **In Bone**, the commonest site.—(*a*) Under the cartilage of the head; (*b*) In the head near the epiphysial cartilage; (*c*) In the neck near the epiphysial cartilage; (*d*) In the acetabulum; (*e*) At the junction of either trochanter (rare, and not strictly hip-joint disease).
2. **In Synovial Membrane.**

The Disease Usually Spreads, wherever it begins, to the: Head of the femur—Acetabulum—Synovial membrane.

It Spreads Occasionally: Through the acetabulum to the pelvis. From the neck to the shaft and great trochanter, without involving the joint. From synovial membrane to psoas bursa, and thence into the pelvis.

Ligaments are invaded by tuberculous granulations from the synovial membrane. The ligamentum teres disappears early. The posterior part of the capsule is perforated. The ilio-femoral, or anterior part of the capsule, usually remains, or is the last ligament to give way.

Synovial Membrane.—Is infiltrated by tubercles. Thickened to form a pulpy swelling.

Head of Femur becomes carious and stripped of cartilage (*Fig.* 111), or necroses and forms a sequestrum owing to the obliteration of its blood-supply by:—

1. Tuberculous focus in the neck.
2. Synovial disease occluding the blood-vessels in the neck.
3. Destruction of vessels in the ligamentum teres

Acetabulum becomes carious May be worn away above and posteriorly by pressure. New bone is formed above and behind the carious acetabulum, thus forming the 'travelling acetabulum'.

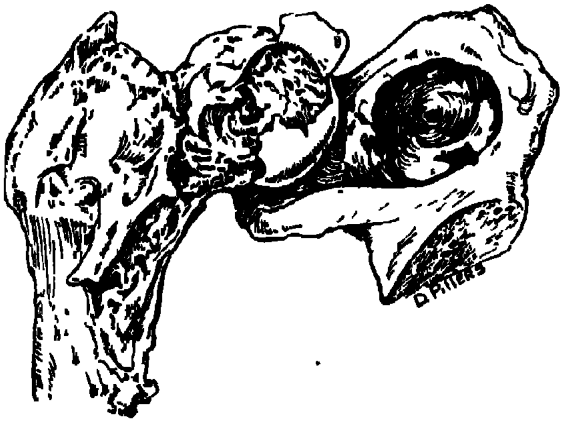

Fig. 111.—Tuberculous hip-joint showing exfoliation of the articular cartilage.

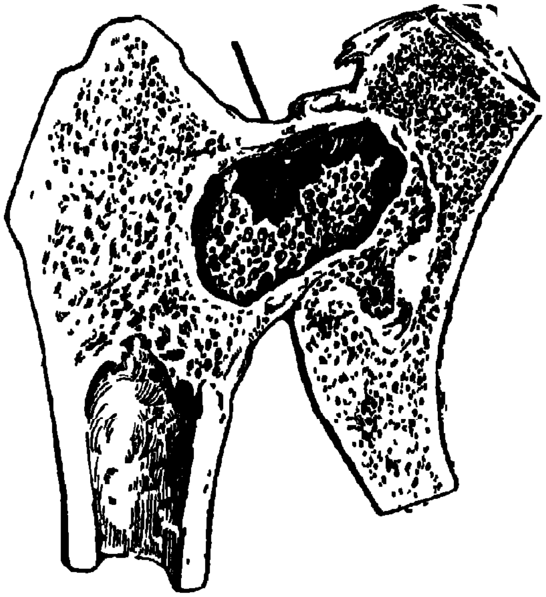

Fig. 112.—Tuberculous hip showing ankylosis. A sequestrum occupies a cavity in the neck of the femur, with a sinus leading from the same.

(*From specimens in the R.C.S. Museum.*)

Tuberculous Disease of Hip—Pathology, ***continued.***

NECK OF FEMUR.—Carious focus may spread: (1) Into the joint; (2) Into the shaft and trochanter outside the joint. May form a chronic tuberculous abscess.

JOINT BECOMES ANKYLOSED (*Fig.* 112):—

By fibrous ankylosis due to the union of granulating surfaces, with subsequent fibrosis.

By true bony ankylosis due to union of carious bony surfaces, with subsequent ossification, if secondarily infected.

JOINT BECOMES DISLOCATED: By destruction of the acetabulum; By destruction of the head or neck; By solution of the ligaments; By contraction of the muscles.

SYMPTOMS.—

FIRST STAGE (stage of synovitis).—Pain in hip, or in knee due to common nerve-supply. Wasting of the thigh muscles. Fullness of inguinal region. Obliteration of the gluteal fold, with flattened nates

Lameness.—At first only a slight—almost unconscious—limp.

Position of the Leg—Thigh is flexed to relax vertical limb of Y ligament. Abducted to relax oblique limb of Y ligament (*Fig.* 113, B). Everted to relax ischiofemoral ligament, and owing to the weight of the limb when patient is lying. Leg is apparently lengthened.

Position of the Pelvis—Tilted down on diseased side so as to bring abducted thigh parallel to fellow. Hence leg is apparently longer Lordosis to compensate for flexion of the thigh. (*Fig.* 113, C.) Scoliosis due to tilting of pelvis

Objective Tenderness.—Pain on extension, adduction, inversion, due to strain on capsular ligaments and pressure of joint surfaces together.

Pain on 'crowding joint surfaces together' by pressing the heel or the great trochanter

Pain on any passive movement, owing to the rubbing of inflamed synovial surfaces.

Rigidity.—All movements of the joint limited, especially extension, adduction, and inversion.

X Rays.—Show decalcification of bones adjacent to joint.

SECOND STAGE (stage of destruction of cartilage and ligaments).—Pain is increased and nocturnal starting occurs. Rigidity is almost absolute.

Position of Leg.—

Flexion.

Adduction. Voluntary movement having ceased, only tonic muscular action is present. The adductors, being much stronger than the abductors, gain this position. (*Fig.* 113, D.)

Inversion. The posterior part of the capsule has given way, and the short external rotators are infiltrated and weakened. (*Fig.* 113, D.)

Apparent Shortening.

Position of the Pelvis.—Lordosis as above. Tilted up on diseased side so as to bring adducted thigh parallel to its fellow; hence scoliosis and apparent shortening. (*Fig.* 113, E.)

X Rays.—Show loss of joint space. Irregularities in articular surfaces.

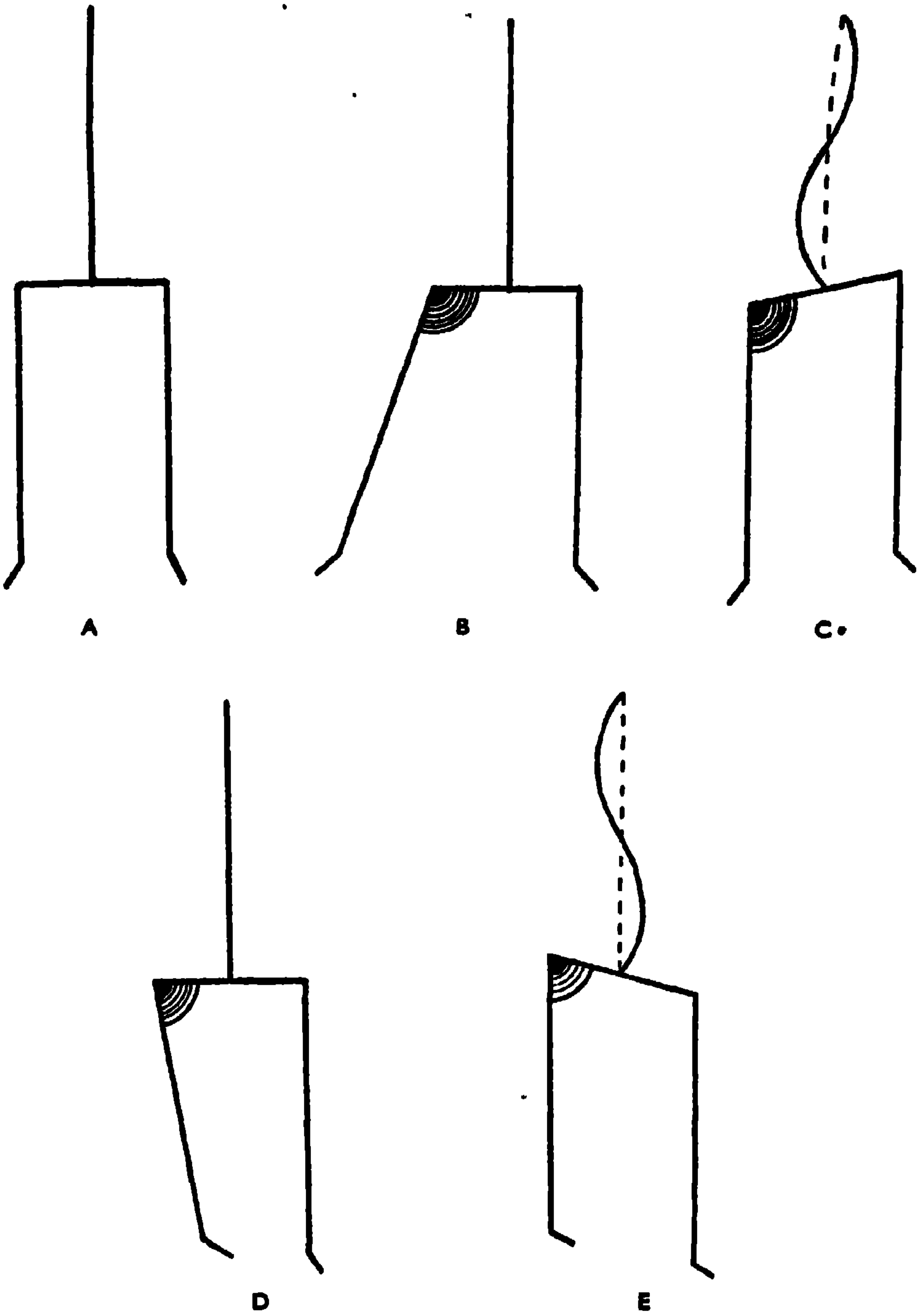

Fig. 113.—Disease or ankylosis of the hip-joint. Diagram showing how the positions of pelvis and spine vary with that of the hip-joint. A, Normal relations. B, Abduction of the hip; position of the leg when the pelvis is level. C, Abduction when the legs are parallel; the pelvis is tilted downwards and the spine curved; the leg is apparently lengthened. D, E, Adduction deformity of the hip; when the legs are parallel the pelvis is tilted upwards and the leg is apparently shortened, while the spine is curved.

Tuberculous Disease of Hip—Symptoms, ***continued.***

THIRD STAGE (Stage of destruction of bone).—Pain as in the second stage. Fever and hectic. Position of the leg as in last in an increased degree, with adduction of—

Actual Shortening due to. (1) Carious destruction of the head; (2) Carious destruction of the acetabulum; (3) Dislocation; (4) Necrosis of the head.

Abscess Formation.—Abscess points· At inner side of great trochanter—Gluteal region—Scarpa's triangle—As a psoas abscess——As intrapelvic abscess bursting into rectum, vagina, or bladder, or ischiorectal fossa—Down the leg towards the knee.

If the abscess bursts and becomes septic, hectic fever and amyloid disease may occur.

X Rays.—Show gross destruction of articular surfaces

DIAGNOSIS.—From ·—

PRIMARY PELVIC ABSCESS.

KNEE-JOINT DISEASE —Knee-joint symptoms.

CARIES OF THE SPINE, PSOAS ABSCESS —Angular curvature, pain, and rigidity of spine

INFLAMED PSOAS BURSA.—Pain only on extension.

SCIATICA.—Nerve tender, flexion hurts more than extension.

SACRO-ILIAC DISEASE.—Compression of pelvis is painful—Apparent lengthening of leg—Pain over sacro-iliac joint.

(In all the above, true hip-joint movements are unimpaired.)

PERTHES' DISEASE —Pain if trivial. X rays show fragmentation of head.

CONGENITAL DISLOCATION.—Absence of pain—Often bilateral—Reducibility.

COXA VARA —Eversion is combined with adduction—Great prominence of the trochanter—Absence of tenderness on pressure—Passive movements painless

DIAGNOSIS OF THE STRUCTURE CHIEFLY IMPLICATED IN HIP DISEASE --

IN SYNOVIAL DISEASE passive movements are almost as painful as active. Movement is much more painful than jarring.

IN DISEASE CHIEFLY AFFECTING THE BONES, active movements are much more painful than passive, because they crowd together joint surfaces by the muscular contraction. Jarring the joint is more painful than passive movements

TREATMENT —

CONSERVATIVE TREATMENT is the first line of treatment, until an estimate can be made of how much the joint surfaces are destroyed.

1. *Rest in Bed* with weight extension or on a frame until the flexion and some of the abduction or adduction have been cured. Continued for three to six months.
2. *Ambulatory Fixation.*—Usually by a plaster spica for about six months, followed by a leather or celluloid splint for about two years. Patient walks on crutches, using a high boot or patten on sound leg.
3. *Extra-Articular Arthrodesis.*—The outer aspect of the joint is exposed and a graft cut from the upper end of the shaft and trochanter. This is fixed along the outer side of the joint so as to produce ankylosis. Or a bone flap is turned down from the ilium and fixed to the trochanter (*Fig.* 114).

Choice between (2) *and* (3).—In purely synovial cases, when after six to twelve months the joint structure is undestroyed, then conservative treatment should be pursued. But if the joint surfaces are destroyed, then ankylosis should be aimed at, and this will be made quicker and sounder by arthrodesis.

OSTEOTOMY for late cases in which ankylosis has occurred in faulty position. (*See below*).

EXCISION is indicated in the third stage of the disease, i.e.:—

When abscesses have formed which, on opening, are found to communicate with the joint.

When marked shortening indicates joint destruction.

Also when prolonged conservative measures, including extra-articular arthrodesis, have failed to cure.

The Anterior Incision outside the sartorius is the most generally useful, because it divides no important blood-vessels, it affords ready access to the joint, and leaves an anterior scar.

The Posterior Incision is most useful when posterior abscesses have formed, or when the head of the bone lies dislocated backwards.

The Removal of all Diseased Structures, especially sequestra in the head and neck of the bone and the acetabulum, is best done by a large flushing gouge.

After-treatment.—The limb should be put up in marked abduction: (1) In order to cause apposition of the stump of the femur and the acetabulum; and (2) To compensate for the shortened limb by the subsequent pelvic tilting downwards on the affected side. An adductor tenotomy may be necessary to bring this about. The limb must be kept abducted for six weeks by a plaster or other splint.

AMPUTATION is very rarely called for. It is indicated when the knee as well as the hip is involved, when other operations have repeatedly failed, when intrapelvic disease is extensive, and when septic sinuses defy other treatment, or when the limb is painful and useless.

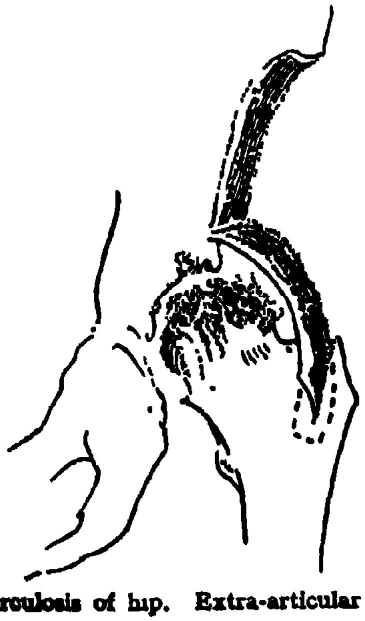

Fig. 114.—Tuberculosis of hip. Extra-articular arthrodesis.

Hip-Joint, Diseases of, ***continued.***

Osteo-arthritis of Hip.—Often traumatic and monarticular. Painful and crippling.

TREATMENT.—By arthrodesis. The articular surfaces are removed and the joint fixed by a long Smith-Petersen nail driven up through the neck and head of the femur into the ilium.

Ankylosis of the Hip.—

CAUSES.—Tuberculosis, pyæmia, gonorrhœa, osteo-arthritis, or trauma.

POSITION.—If no preventive treatment has been employed, flexion, adduction, and inversion will be marked

TREATMENT.—Nothing is required if the limb is in good position without much shortening

Gradual correction by weight extension for false or fibrous ankylosis in a bad position.

Fixation in an abducted position (*see* AFTER-TREATMENT OF EXCISION OF THE HIP FOR TUBERCULOUS DISEASE, *above*) is very valuable in cases with much shortening.

Arthroplasty, in young and vigorous subjects. A U-shaped flap is turned up from the outer side of the trochanteric region, the trochanter sawn off, the joint exposed, and chiselled until the femur is free. Bone is removed from the femoral head and from the acetabulum. The socket is lined by a flap of fascia lata. The limb is moved directly after the skin incision has healed

Osteotomy Customary to use a Lorenz bifurcation osteotomy (*see* p. 201 and *Fig.* 71) or a McMurray subtrochanteric osteotomy.

KNEE-JOINT

Incidence of Disease.—The knee-joint is more commonly affected by injury and disease in all its varieties (except perhaps gout) than any other joint.

Simple Synovitis.—The joint is held in a semi-flexed position.

Swelling appears at the sides of the patella, above and below at first, and then it assumes a horseshoe shape, surrounding the patella, except below in the position of the patellar ligament.

Fluctuation can be obtained from above to below the patella, pressing on the sides of the swelling.

The patella can be made to tap against the femoral condyles. This is best done when the limb is extended and the quadriceps is relaxed.

Tuberculous Disease (*Fig.* 115) begins most commonly in the bones in children, and in the synovial membrane in adults.

Rarely bone disease may produce an extra-articular abscess without affecting the joint.

IN THE FIRST STAGE of disease of the joint cavity, the signs of synovial distension as above given are present; but the hollows of the knee are filled by a doughy substance instead of fluid.

Later, especially in children, a spindle-shaped swelling is formed by the knee, the muscles of the thigh and calf being much wasted.

X-ray in children may show an increase in growth activity in the tibia of the affected side due to stimulation of the epiphysial cartilage. Also the patella of the affected side ossifies more quickly than the non-affected side.

IN THE SECOND STAGE, displacement occurs, which in its complete form is fourfold, i.e., displacement of the tibia backwards and outwards, flexion, and external rotation. This is due to the predominant action of the hamstring muscles, and especially the biceps.

THIRD STAGE.—Actual shortening of either femur or tibia, or of both, from absorption, is produced by bone destruction.

Abscesses form, and usually point at the side of the patella, but may track up the thigh or down the leg.

TREATMENT.—The knee-joint probably gives worse results than any other after conservative treatment, i.e., the majority of cases relapse after it, and come to an eventual operation.

CONSERVATIVE TREATMENT.—The limb is, if necessary, straightened by a weight extension, and then fixed in a Thomas's knee splint. Bier's treatment may be well combined with this. Probably six to twelve months will be required, but much shorter periods are usually employed. It is indicated as a first measure in all early cases, especially in children; and in cases when the disease is purely osseous or purely synovial.

EXCISION is indicated: (1) When conservative measures have failed; (2) When the disease has progressed to the second and third stages; (3) In almost all adult cases; (4) When faulty ankylosis has occurred.

Bony ankylosis in a straight position should be aimed at. The lateral and crucial ligaments are freely divided, and all the synovial membrane and cartilages removed, with all the diseased bone. • A stout graft is cut from the tibia and slid upwards to join the femur (*Fig.* 116).

After excision in children it is essential that a long case splint, from the gluteal fold to the ankle, should be worn until bone growth has ceased. Otherwise bending will probably occur at the site of excision.

AMPUTATION is very rarely called for. Septic sinuses with necrosis at the back of the joint may demand it.

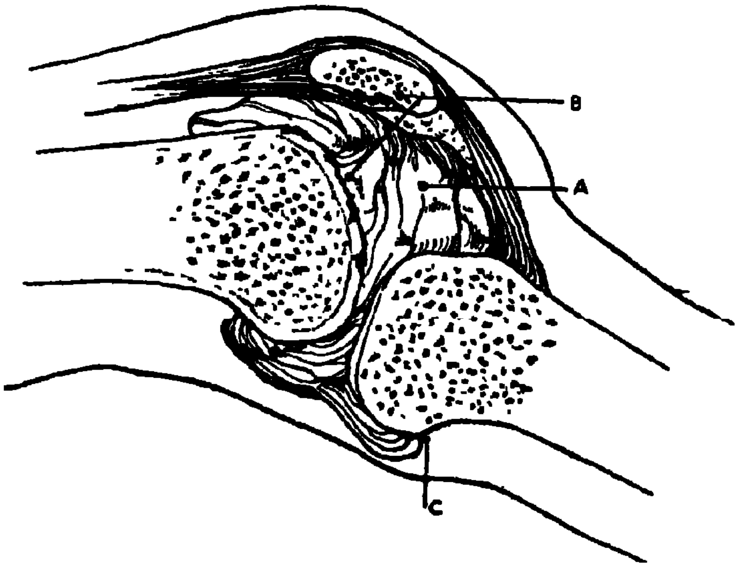

Fig. 115.—Tuberculous disease of the knee-joint, viewed in section. The tibia is dislocated backwards. A, Diseased synovial membrane; B, Articular surfaces eroded; C, Extension of synovial disease downwards and backwards into the leg.

Tuberculous Disease of the Knee-Joint—Treatment, *continued.*

Ankylosis usually results from septic or tuberculous arthritis.

Flexion with backward and outward displacement of the tibia and eversion of the leg is the common position. Hyperextension is occasionally seen.

TREATMENT.—Weight extension, aided by hamstring tenotomies in early fibrous ankylosis. Excision and fixation in bad fibrous and in bony ankylosis Cuneiform or curved osteotomy and rectification in bony ankylosis (*cf. Fig.* 72, p 202).

ANKLE-JOINT

Synovial Distension is rare from simple causes. The characters are similar to the early stages of tuberculous disease.

Tuberculous Disease begins in the astragalus, or less commonly in the synovial membrane or tibia or fibula. The swelling appears under the extensor tendons in front of the joint, and at the sides of the tendo Achillis behind. The foot is held in a position of plantar flexion.

The disease is very liable to spread to the other tarsal joints.

TREATMENT —Conservative measures should be given a prolonged trial.

Scraping out diseased foci through anterior incisions, with REMOVAL OF THE ASTRAGALUS, is required in relapsing or suppurating cases

AMPUTATION is indicated when other measures have failed, and especially in older patients.

Ankylosis.—This is very common as the result of injury or talipes If in faulty position an astragalectomy will often enable it to be rectified

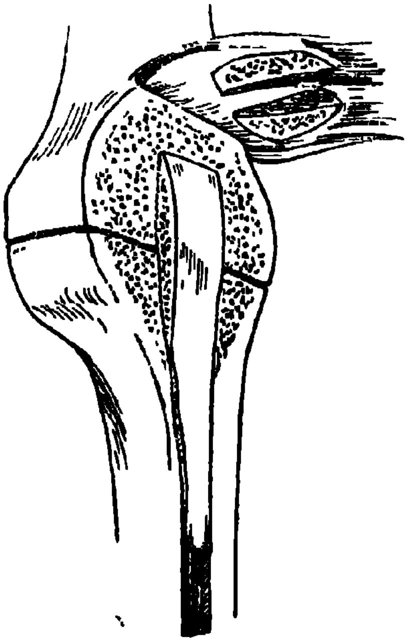

***Fig.* 116.—Tuberculous knee. Excision of joint. A portion of the anterior border of the tibia has been slid up into the femur. The patella has been denuded of cartilage and divided into two portions. These will also lie over the line of union.**

TARSAL JOINTS

Incidence of Disease.—Gonorrhœa, tubercle, osteo-arthritis, and gout are the commonest affections, together with the changes secondary to carious forms of talipes.

Tuberculous Disease.—Bones are often primarily affected, especially the os calcis, astragalus, scaphoid, and cuboid.

The joints between the astragalus and scaphoid, the three cuneiforms, and the three inner metatarsals communicate with one another so easily that disease of one generally spreads to the others.

The joints between the os calcis and cuboid, and between the cuboid and the two outer metatarsals, may be affected singly.

Swelling occurs over the affected joints, and is diffuse. Tenderness is marked over affected bones.

X rays show great decalcification of all the bones of the foot. Later, destruction of joints.

TREATMENT.—

CONSERVATIVE TREATMENT should be tried for prolonged periods.

LIMITED OPERATIONS are suitable only in rare cases when one bone or joint is alone affected, e g., the astragalus or os calcis, or calcaneo-cuboid joint.

AMPUTATION by Syme's method, or at site of election, is usually required in cases where other methods have failed, when septic sinuses have arisen, and when the patient cannot afford long conservative treatment

CHAPTER XXIV

INJURIES AND DISEASES OF THE SPINE

FRACTURES OF THE SPINE

Causes.—

DIRECT VIOLENCE.—Spine breaks at the point struck.

INDIRECT VIOLENCE.—Spine usually breaks at about the junction between the most movable and the most fixed parts, i.e., between the neck and back, or between the thorax and lumbar region.

Varieties.—

INCOMPLETE FRACTURES —The continuity of the column is not destroyed.

Spinous processes, transverse processes, and laminæ may be broken and displaced. They are of importance because late affections of the cord may follow, and also because they may cause pain in the back and inability to work Such conditions must be sought for in all cases of chronic back sprain.

COMPLETE FRACTURES or fracture-dislocations

COMPRESSION FRACTURES —One or more bodies and discs are crushed

Fracture-dislocations.—

ANATOMY.—Most common in the cervical and upper dorsal regions, also in the last dorsal and first lumbar vertebræ The articular processes are usually fractured, except occasionally in the cervical and lumbar regions. The body or intervertebral disc is fractured or torn The ligaments are extensively lacerated. Fracture, comminution, and impaction of the laminæ and spinous processes may occur The upper fragment is usually displaced forward.

The cord is either crushed between the laminæ above and the bodies below, or it is cut across or transfixed by bony fragments, or its structural continuity is severed, whilst the bones regain their normal position by natural recoil or treatment

SIGNS.—

DEFORMITY OF THE SPINE.—Usually an angular curvature. There may be a mere irregularity in the line of the spinous processes.

SIGNS OF LOCAL TRAUMA —Bruising, pain, swelling.

PARAPLEGIA, ANÆSTHESIA, LOSS OF THE REFLEXES (*see* p 279).

VASOMOTOR PARALYSIS, with rise of temperature in the parts below the lesion up to 110° F., or even higher.

TROPHIC LESIONS.—

1. Acute bedsores form over the sacrum or buttocks. Less often over the anterior superior spine or heels. They occur about three days after the injury, or at any later date if myelitis arises.

Pressure, and fouling with urine or fæces, act as exciting causes. A large deep slough forms rapidly, and finally the bone is exposed.

2. Of the urinary organs. Sloughing may occur in the bladder, urethra, penis, or scrotum. Septic inflammation ascends to the kidney as a pyelitis and pyelonephritis.

AFFECTION OF THE BLADDER MUSCLES.—Either true incontinence, retention with overflow, or involuntary reflex micturition.

PARALYSIS OF THE RECTUM or its sphincters.

METEORISM from the abdominal-wall paralysis.

PRIAPISM.

EMBARRASSMENT OF RESPIRATION by the paralysis of the abdominal muscles and the intercostals.

TREATMENT.—

GENERAL.—To be lifted upon some supporting sheet, and not picked up by the legs and shoulders. In all cases the patient should be carried in a prone position, i.e., face downwards; this tends to undo the dislocation or compression, whereas the reverse position makes it worse Bed should be firm and upon fracture boards. Special care of the lower extremities lest they get burned by hot-water bottles.

REDUCTION under an anæsthetic —If the lesion is below the cervical region; if the paraplegia is incomplete. Is best performed about twenty-four hours after the accident, when shock has passed off.

LAMINECTOMY.—(1) In partial lesions of the cord, indicated by retention of deep reflexes and of the senses of heat and pain. (2) In cases where dislocation cannot be reduced or cannot be retained and where the cord is uninjured. These conditions may occur in the cervical or lumbar regions.

SYMPTOMATIC TREATMENT.—(*a*) Alone: when total transverse lesion has occurred. (*b*) In conjunction with or following any of the above: Retention apparatus, e g , sand-bags Patient should be moved as a whole and not twisted. Skin kept dry and clean with spirit lotions and antiseptic powders. The sheets kept smooth and clean. The urethra disinfected and kept covered by an antiseptic dressing. The urine drawn off by sterile rubber catheter where vesical paralysis exists.

Special spinal bed, turned daily (*Fig.* 117).

Impaction of Dislocated Articular Processes.—Only occurs in the lumbar region and after great violence. Can only be recognized by good anteroposterior radiographs Reductions cannot be effected by usual hypertension.

TREATMENT.—Open operation and leverage of displaced articular process (after flexion of spine). Generally it is necessary to remove one of the impacted articular processes (*Fig.* 118).

Compression Fractures.—

ANATOMY.—Usually found at the junction of the dorsal and lumbar spine, from the 11th dorsal to the 2nd lumbar vertebræ. Caused by violent flexion forwards of the body. One or two of the bodies and discs are crushed together, and appear to be wedge-shaped when viewed laterally. (*Figs.* 119, 120.)

Compression Fractures of the Spine—Anatomy, ***continued.***

KÜMMEL'S DISEASE is a variety of this injury in which the original accident is comparatively slight and in which the symptoms do not appear for some months after the patient has been getting about. Possibly in this disease the original injury is one to the blood-vessels, causing bone atrophy with subsequent yielding of the soft bone under the body weight.

SYMPTOMS AND SIGNS.—Pain and weakness in the back. There is prominence of one vertebral spine which corresponds to the area of maximum tenderness. Often there is exaggeration of the knee-jerks and marked neurasthenia. Lateral X rays show one or two vertebral bodies to be wedge-shaped, and the discs damaged or distorted.

TREATMENT.—This should be carried out at once, before fixation of the deformity has had time to occur and before neurasthenia develops.

1. HYPEREXTENSION WITH PLASTER —Under local or general anæsthesia. The patient is slung face downwards, and the spine forcibly hyperextended (*Fig.* 121). Fixed by a plaster jacket in this position.
   After-treatment —After 3–5 days the patient gets up and has graduated exercises, e g., carrying weights on the head, flexion of hips, and lifting weights with the abdominal muscles. Plaster jacket is retained for three to four months and may be followed by the use of a spinal brace. (*Fig.* 122)
2. SPINAL BRACE.—Used for patients unsuited for, or refusing operation, and as the after-treatment of either of the above.

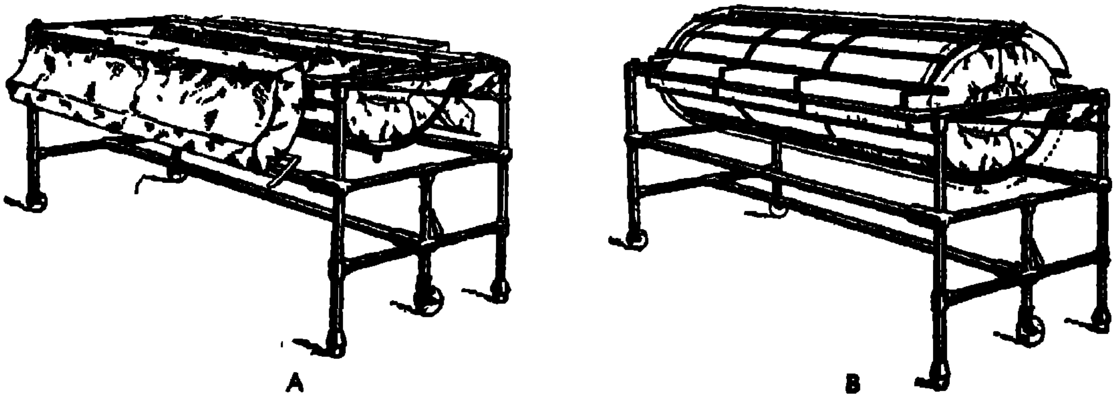
A
B

Fig. 117.—Hey Groves's improved spinal bed. A, Open position of bed—patient lies in the trough; B, Closed position of bed, ready for turning.

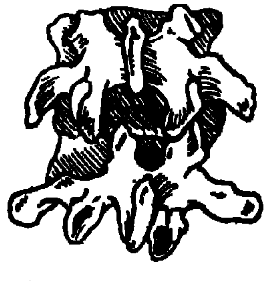

Fig. 118.—Dislocation of the cervical spine, showing impacted articular process.

DISLOCATIONS OF THE SPINE

OCCUR only in the cervical region, where the horizontal articular surfaces allow this possibility; most commonly between the 5th and 6th cervical vertebræ

VARIETIES.—Unilateral and bilateral

DISPLACEMENT—The upper part of the spine is displaced forwards, the upper articular processes locking against the lower.

SYMPTOMS AND SIGNS.—Sudden death in complete dislocations in the upper part. Signs of cord injury in most cases Neuralgia of the nerves at the seat of injury. Some deformity, either forward displacement or rotation.

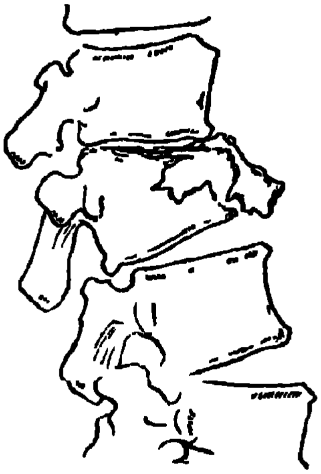

Fig 119.

Fig 119—Compression fracture of 1st lumbar vertebra.

Fig 120—Kümmel's disease affecting the 10th and 11th dorsal vertebræ.

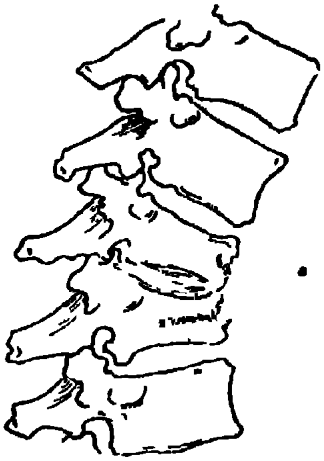

Fig 120.

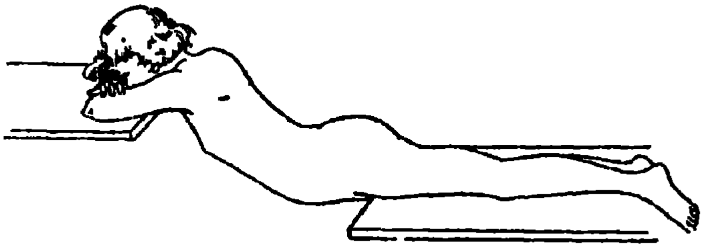

Fig 121

Fig. 121—Fracture of spine. Position of patient in application of plaster.

Fig. 122.—Fracture of spine. Plaster jacket Patient carrying weight.

Fig. 122.

Dislocations of the Spine, *continued.*

TREATMENT.—Reduction under an anæsthetic, or open operation when reduction has failed, with, possibly, removal or part of the lower articular processes. Articular processes when reduced must be fixed to prevent redislocation.

DISEASES OF THE SPINAL CORD AND NERVES WHICH MAY FOLLOW INJURIES

Varieties.—(1) Traumatic neurasthenia (railway spine); (2) Concussion of the cord (molecular disintegration), (3) Spinal meningitis; (4) Hæmorrhage into or around the cord; (5) Myelitis; (6) Laceration of the cord; (7) Paraplegia.

1. **Traumatic Neurasthenia** (railway spine) occurs after a severe accident which has caused both mental and physical shock.

THE SYMPTOMS are usually absent or trivial for the first few days after the accident. Pain in the head and back, especially in the lumbar region. Increased knee-jerks. Mental changes: a loss of the power of concentration, undue excitability, etc Some functional disturbance of micturition. Varying and changing areas of partial anæsthesia or hyperæsthesia.

TREATMENT is constitutional—chiefly rest. Supervised rehabilitation is necessary

2. **Concussion of the Cord.**—A molecular injury or disintegration without visible lesion.

It produces exactly the same symptoms as a partial or total laceration at the time, but the symptoms (paraplegia) may rapidly disappear. Priapism is said not to occur in concussion. But more frequently the symptoms are permanent.

3. **Spinal Meningitis** may be simple and plastic, or septic.

Localized pain in the back increased by movement Neuralgic pain along the course of the spinal nerves. Spastic rigidity, painful cramps. Hyperæsthesia, exaggerated reflexes.

Meningitis serosa circumscripta may develop and lead to the formation of a subdural cyst. Laminectomy and removal of the cyst may be necessary.

4. **Spinal Hæmorrhage.**—

a. INTRAMEDULLARY.—Hæmorrhage into the central parts of the cord. Most common in the lower cervical region.

Paraplegia of an incomplete type immediately follows the injury. Senses of pain and temperature are often lost without anæsthesia. No signs of irritation of the spinal nerves. Permanent damage caused by spinal degeneration.

b. EXTRAMEDULLARY.—Hæmorrhage between the bones and dura, and between the dura and cord.

Paralysis comes on gradually at an interval after the injury.

Severe pain with muscular spasms is caused by irritation of the roots of the spinal nerves.

Recovery may be expected, as no permanent cord damage results.

5. **Myelitis** may arise from a septic wound or as a part of the result of any spinal injury.
   The usual symptoms of paraplegia are associated with specially marked trophic disturbances, e.g., acute bedsores, or sloughing cystitis; rapid atrophy of muscles supplied by the affected segments.

6. **Laceration of the Cord.**—Paraplegia is the most constant and earliest result. Meningitis or myelitis often complicates the paraplegia. Spinal hæmorrhage may accompany or may cause the paraplegia.

7. **Paraplegia** resulting from spinal injuries.—
   RELATION OF THE CORD TO THE VERTEBRAL SPINES:—
   Fourth cervical nerve segment (diaphragm) corresponds to 2nd cervical spine.
   The cervical enlargement (arm) to 3rd-7th cervical spines.
   The lumbar enlargement to 10th–12th dorsal spines.
   The lumbar segments (front of the thigh, etc) to 9th–11th dorsal spines.
   The sacral segments (the rest of the leg, etc , rectum and bladder) to 11th dorsal–1st lumbar spines.
   Cauda equina to 2nd lumbar–2nd sacral spines.
   CAUSES OF TRAUMATIC (SPINAL) PARAPLEGIA —
   1 IMMEDIATELY following the injury —Concussion (without manifest injury). Contusion with intramedullary hæmorrhage. Laceration of the cord by crushing or penetrating wounds.
   2. ARISING AFTER AN INTERVAL —Extramedullary hæmorrhage (twenty-four to forty-eight hours after, without pyrexia). Inflammatory exudation of meningitis (three days or more after, with pyrexia). Pressure of callus or cicatrices (two weeks or more after)
   SIGNS OF A TOTAL TRANSVERSE LESION.—
   1 IN THE AREA SUPPLIED BY THE NERVES BELOW THE LESION —Complete motor paralysis, followed by late rigidity and contraction of the muscles Complete anæsthesia, with loss of senses of pain or temperature. Complete and permanent loss of the deep reflexes Temporary loss of the superficial reflexes. Vasomotor paralysis, with trophic lesions. Visceral paralysis (bladder, rectum, intestines) according to the site
   2. IN THE AREA SUPPLIED BY THE NERVES AT THE SITE OF THE LESION. —Paralysis, with rapid and flaccid atrophy of the muscles. Zone of hyperæsthesia from nerve-root irritation.
   SIGNS OF A PARTIAL LESION.—The central parts of the cord may excape injury In this case the deep reflexes are retained or soon regained; the senses of pain and of temperature are retained; the anæsthesia may be only partial.
   In cases of recovery from concussion or contusion the functions of the cord are regained in the following order: Deep reflexes—Sense of pain and temperature—Tactile sensation—Motor power.

Special Features of lesions occurring at different levels of the spine.—

1. LOWER LUMBAR AND SACRAL REGIONS.—The cauda equina is crushed.
   Paralysis of all leg muscles except the psoas, iliacus, quadriceps extensor femoris, and the adductors.
   Paralysis of all the perineal and penile muscles.

Diseases of Spinal Cord following Injuries—Special Features, *continued.*

Anæsthesia of the perineal and genital regions, also of the foot and the outer side and back of the leg below the knee.

Bladder is paralysed. Retention at first with overflow, but true incontinence later from atrophy and relaxation of the sphincters.

Rectum: The sphincter is paralysed, and incontinence of fæces results.

2. LOWER DORSAL AND UPPER LUMBAR REGIONS.—The lumbar enlargement is injured, with the origins of the lumbar and sacral plexuses, and the centres for the bladder and rectum.

   Total paralysis and anæsthesia of the legs and perineum.

   Bladder and rectum, from an injury to their centres, have their sphincters relaxed from the first, with true incontinence of urine and fæces.

   Marked tendency to sloughing of the skin and bladder.

3. MID-DORSAL REGION.—In addition to the paralysis and anæsthesia in the last, paralysis and anæsthesia of the abdominal wall and some of the lower intercostal spaces.

   Meteorism results from the abdominal paralysis.

   A painful girdle at the waist occurs from hyperæsthesia.

   Bladder shows retention with overflow, but later a reflex involuntary micturition may occur.

   Rectum is not much affected, but constipation is marked from the abdominal paralysis.

4. UPPER DORSAL REGION.—In addition to the above, paralysis of the intercostal muscles.

   Bronchitis with congestive pneumonia is very frequent.

   Priapism may occur.

5. LOWER CERVICAL REGION.—In addition to the last, paralysis and anæsthesia of the arms.

   Priapism is almost constant.

   The pupils are narrowly contracted, forming the so-called 'spinal miosis', from a cutting of the sympathetic fibres to the pupil, which descend in the cord and then leave by the lower cervical and upper dorsal nerves.

   SEVENTH CERVICAL SEGMENT.—Hands half closed. Elbows bent and forearms pronated over chest.

   SIXTH CERVICAL SEGMENT.—Arms rolled out and abducted. Elbows flexed. Hands supinated. Fingers semiflexed.

   FIFTH CERVICAL SEGMENT.—Arms paralysed and by side of trunk.

6. AT OR ABOVE THE THIRD CERVICAL.—Death from asphyxia, due to involvement of the phrenic (fourth cervical) and the intercostal nerves.

TUMOURS OF THE SPINAL CORD

Position.—Meningeal (66 per cent). Extradural (20 per cent). Intramedullary (14 per cent).

Nature.—

1. MENINGEAL.—Meningioma—hard and localized, or soft and spreading.
2. EXTRADURAL.—Sarcoma. Lipoma. Fibroma. Angioma.
3. INTRAMEDULLARY.—Glioma. Granuloma, e.g., tuberculoma.

Symptoms are very slow and insidious, and therefore most cases are overlooked and mistaken for spastic paralysis or disseminated sclerosis. In general, the symptoms are:—

IN EXTRADURAL TUMOURS the pressure is exerted first on the nerve root of one side and then on the other half of the cord, giving root pain, unilateral cord compression, and finally general cord compression.

INTRADURAL EXTRAMEDULLARY SWELLINGS give prolonged root pressure and then slow-growing compression of cord.

INTRAMEDULLARY TUMOURS give no root pain, but a partial paraplegia from the onset.

PAIN, chiefly marked at the part corresponding to the spinal segment, e.g., in the arms in a lower cervical tumour. Most marked in extradural and meningeal growths.

PARÆSTHESIA as a band or girdle—when the posterior roots are irritated—affords the best localizing evidence.

PARALYSIS WITH WASTING in the parts supplied by the segments affected.

PARALYSIS WITH SPASM in the parts below the lesion. Thus spastic paralysis of the legs with exaggerated reflexes is present in nearly all cases.

ANÆSTHESIA of variable extent and degree.

VISCERAL PARALYSIS of bladder and rectum are late symptoms.

Diagnosis is often very difficult. The association of an area of pain above (corresponding to the lesion) with an area of spastic paralysis below is the most significant feature The injection of lipiodol through the occipito-atlantal membrane and subsequent X-ray examination will locate a tumour if it is obstructing the dural canal

Treatment.—Removal.

LAMINECTOMY of several vertebræ. The mistake is usually made of exploring too low.

OPENING THE DURA —If the growth is intradural it may be removed. If intraspinal, the cord is divided longitudinally over the growth and the wound temporarily closed

SECOND STAGE.—A week later the wound is opened and the tumour is often found to have extruded itself upon the surface.

CLOSURE OF THE WOUND.—The dura, muscles, and skin are very carefully closed in separate layers

SPINA BIFIDA

Definition.—A deficiency of the vertebral neural arches, through which a tumour formed by mal-developed cord or membranes protrudes.

Varieties (*Fig.* 123).—

MYELOCELE.—Central canal of the cord opens upon the skin surface in the sacral region (incompatible with life).

SYRINGOMYELOCELE.—Central canal is dilated, and the nerves pass outside the cyst.

MENINGOMYELOCELE.—Dilatation of the membranes forms a cyst; the cord is attached to its dorsal wall, and the nerves pass through the cyst.

MENINGOCELE.—A cyst formed of spinal membranes posterior to the cord, which is not affected.

SPINA BIFIDA OCCULTA.—No tumour occurs, except perhaps a lipoma or a dermoid with long hair.

Spina Bifida, *continued.*

Signs.—

Tumour in the median line of the spine. Usually over the lumbo-sacral region, rarely in the cervical or dorsal The tumour is (except in spina bifida occulta) translucent and fluctuating.

Pressure on the Tumour produces bulging of the anterior fontanelle, and convulsions.

The Edges of the Gap in the vertebral laminæ can be felt.

The Skin Covering the Tumour is thin, red, often covered with dilated vessels or ulcerated.

Meningomyeloceles and syringomyeloceles are accompanied by spastic paralysis of leg muscles, talipes, pes cavus, and interference with bowel and bladder mechanism

Complications.—These are most often found in the syringomyelocele.

Other congenital defects—hydrocephalus, talipes. Trophic ulcers, perforating ulcer Arthropathies (Charcot's joint). Ankylosis of the small joints of toes Various forms of paraplegia.

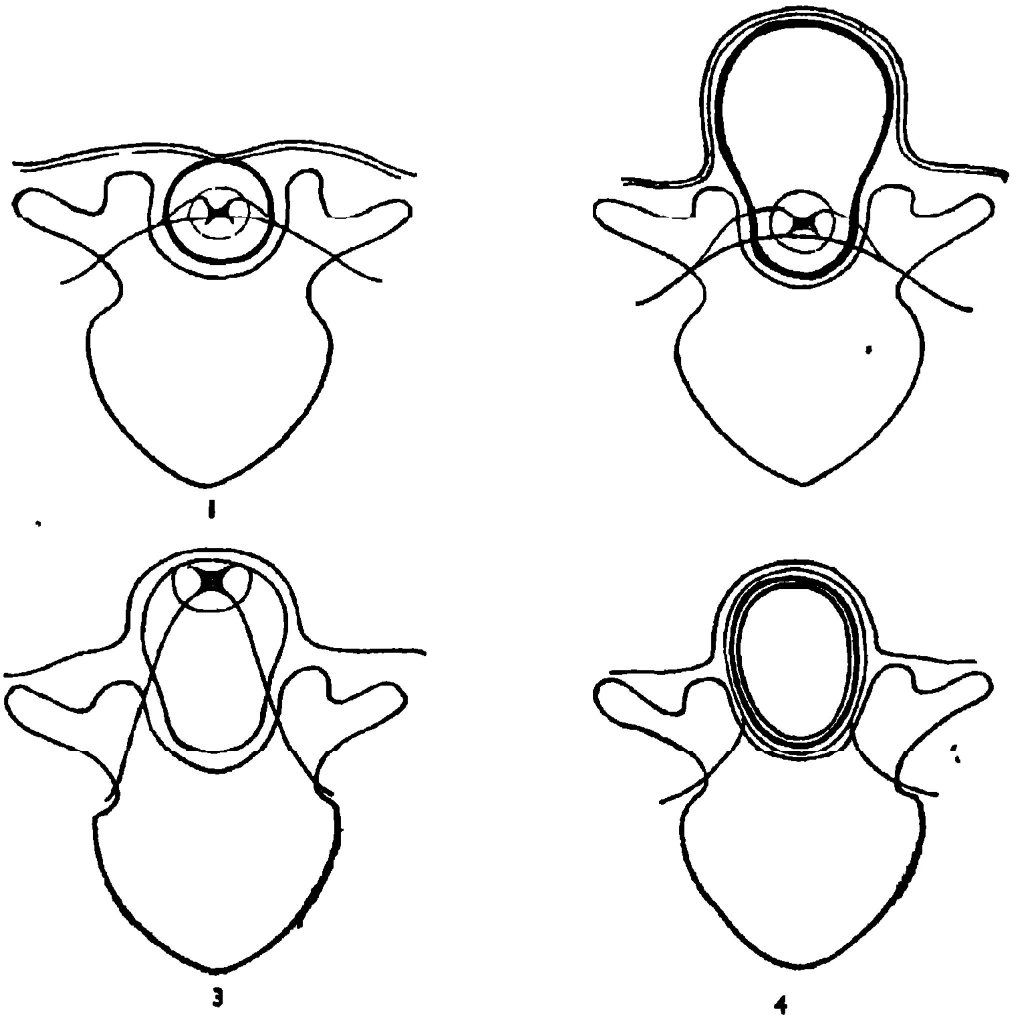

Fig. 123.—Varieties of spina bifida. 1, Spina bifida occulta, with normal cord, 2, Meningocele; 3, Meningomyelocele; 4, Syringomyelocele.

Prognosis.—Usually very bad. It is serious according to the variety in the order named above. The thickness and nutrition of the overlying skin form another great factor in prognosis.

Treatment.—

MERE PROTECTION by a pad if the skin is thick and healthy

PUNCTURE.

OPERATION, with an attempt to replace the cord and nerves and sew the membrane and skin over them.

The patient is placed in an inverted position to lessen the escape of fluid. The prospect of success is very slight, except in pure meningoceles, which are rare.

REMOVAL OF A LIPOMA OR DERMOID in spina bifida occulta when it is causing paraplegia; often very successful.

SACRO-COCCYGEAL TUMOURS

Origin.—From non-closure or overgrowth of the neurenteric canal.

Varieties.—Dermoid cyst (in connexion with the posterior rectal wall; between the rectum and coccyx, on the skin surface over the coccyx or sacrum) Composite tumours chiefly are · Myxomatous adenoma (probably this is really a teratoma) Lipoma (probably a degenerate dermoid). Teratoma or partially developed twin. Cystic hygroma Sarcoma. Chordoma.

Treatment.—Removal by dissection.

If this has to be done in infancy the results are very bad.

TUBERCULOUS DISEASE OF THE SPINE

(*Pott's Disease*)

Aetiology.—Children most commonly, but it may occur at any age. A strain or blow may act as a predisposing cause.

Anatomy.—Lower dorsal region is the common site. Cervical region rarely affected, except in children. Lumbar region rarely affected, except in adults. There are three different anatomical types·—

1. PERIOSTEAL.—The disease begins beneath the periosteum of the bodies, beneath the anterior common ligament. It spreads beneath the ligament from body to body, and so affects a number of vertebræ. It invades the regions of the intervertebral discs.
   It is specially common in adults
   It produces no angle, but only a slight kyphotic bend.
2. CENTRAL.—The disease begins in the body at the point at which the thin epiphysis joins it. It spreads into the centrum, the adjacent disc, and thence to the next body, producing caries and softening. The weight of the column above compresses and crushes the soft carious bodies, and produces an angular deformity. This form thus attacks a few (usually two) vertebræ only (*Fig.* 124).
   It is the common type in children.
3. LOCALIZED DISEASE limited to one vertebra.—Usually in the lumbar region Either under the anterior common ligament, or affecting one of the processes. A definite sequestrum may be formed.
   No deformity and very few symptoms unless an abscess result.

After its origin, CASEATION, SUPPURATION, or NECROSIS (rarely) takes place.

Pott's Disease—Anatomy, *continued.*

THE DISEASE SPREADS by abscess formation (*see* p. 285), or more rarely to the spinal membranes, producing a local thickening, with pressure on, or disease of, the cord.

CURE TAKES PLACE by a falling together of healthy bones so as to obliterate the diseased focus.

ANKYLOSIS, with obliteration of the affected joints, is the last stage

Signs and Symptoms.—

1. PAIN.—

a. LOCAL PAIN at the site of the disease Often inconspicuous, but it may be a constant dull ache.

Is produced by jarring the spinous processes; by pressure on the head or jerks on the feet; by pressure on the transverse processes; by flexion or rotation of the spine.

b. REFERRED PAIN, produced by pressure on nerve-roots Neuralgia of any of the spinal nerves, according to the site of the disease.

Intercostal neuralgia, or an abdominal girdle pain, are much the most frequent of these signs. In children a stomach-ache may be the only complaint

2. RIGIDITY —Muscular rigidity in the early stages of the disease, the muscles being held rigid in order to prevent painful movement. Ankylosis produces absolute and painless rigidity later The spine cannot be flexed or rotated: this is most marked in the upper regions of the spine. In the cervical region the head is supported by the hands

In little children, place the patient on his face and lift the feet the natural dorsiflexion which would be produced is prevented.

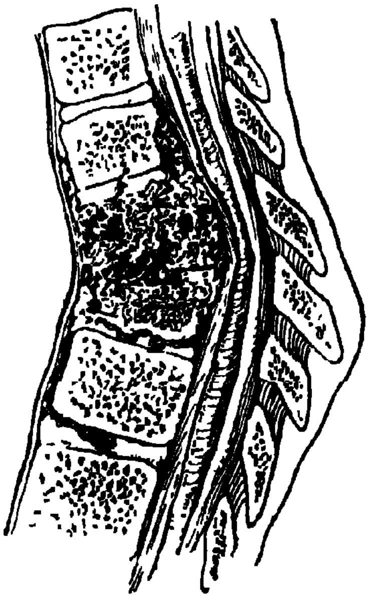

Fig. 124.—Caries of the spine. Disease starts in the body of the vertebra, destroys the intervertebral discs, spreads down the anterior common ligament, and backwards towards the cord.

3. DEFORMITY.—Angular displacement at the site of the disease is the rule in 'central' disease The bodies fall together, and the spinous process of the vertebra above (dorsal region) or of the vertebra below (lumbar region) forms an angle projecting backwards.

A backward curve affecting several or many vertebræ usually results from the periosteal type of disease.

No displacement at all occurs in very limited disease or in extensive disease which is quite superficial, and it is rare in cervical disease.

Compensatory curves in a forward direction occur above and below the disease in order to maintain the equilibrium of the body

In the typical extreme angular deformity of the dorsal region, a hump is produced by it; the head is sunk low between the shoulders; the sternum is bent forwards, the ribs are crowded together, and the heart is displaced; lordosis exists in the lumbar region.

4 ABSCESS.—A tuberculous suppuration forms beneath the anterior common ligament, and tracks downwards or laterally (*Fig.* 125).

IN THE CERVICAL REGION.—A retropharyngeal abscess. (*a*) Breaks into the pharynx; Spreads into (*b*) The side of the neck; (*c*) The axilla; (*d*) The mediastinum.

IN THE DORSAL REGION the abscess. (*a*) Runs down behind the diaphragm into the psoas sheath, (*b*) Follows the dorsal branch of the intercostal vessels and points outside the erector spinæ; (*c*) Follows the main intercostal vessels and points between the ribs; (*d*) *Very rarely tracks upwards into the neck, (*e*) May remain locally as a paravertebral abscess.

IN THE LUMBAR REGION the abscess: (*a*) Follows the lumbar vessels, and presents outside the erector spinæ or in Petit's triangle; (*b*) Forms an iliac or psoas abscess

(For further details of these abscesses, *see* pp. 6–9)

5. PARAPLEGIA.—

CAUSED BY: (*a*) Pressure of tuberculous material upon the cord, (*b*) An abscess pressing upon the dura mater, or (*c*) Tuberculous disease of the cord or its membranes. In severe angulation in the dorsal region, the posterior aspect of the damaged vertebræ may cause pressure on the cord

Rarely if ever is it the result of a narrowing of the bony canal, which is usually larger, rather than smaller, then normal.

SPECIAL FEATURES.—A zone of painful anæsthesia corresponding to the nerves at the site of disease. Motor weakness of the legs, the toes being dragged and the limbs feeling heavy and painful. Sensation is unaffected at first. The reflexes are exaggerated. Finally, complete paraplegia may result, with anæsthesia, paralysis of the sphincters, and painful involuntary contractions.

RAPID DEATH may occur from a bursting of an abscess through the dura mater producing meningitis.

SUDDEN DEATH may result from disease of the atlas or axis producing dislocation with a crushing of the cord.

Prognosis.—

GOOD as regards the prospect of life in uncomplicated cases.

BAD SIGNS and causes of death are: Abscesses which become septic—Implication of the spinal cord and its membranes—Septic sores and cystitis—Tuberculous involvement of other organs.

Pott's Disease, *continued.*

Diagnosis.—When pain, rigidity, and deformity coexist, diagnosis is easy. Three groups of cases present difficulty, viz.:—

1. CASES WHERE DEFORMITY IS ABSENT, pain being the chief symptom.—

LUMBAGO, SCIATICA, NEURALGIA.—In all these marked tenderness exists on pressure over the affected muscles or nerves—No tenderness or pain on pressing on the bones—Rapid relief of pain by salicylates, etc., is often seen.

SPRAINS OR OTHER INJURIES OF THE SPINE.—In these there occur: Relation to a recent injury—Ecchymosis—Symptoms rapidly clear up with rest.

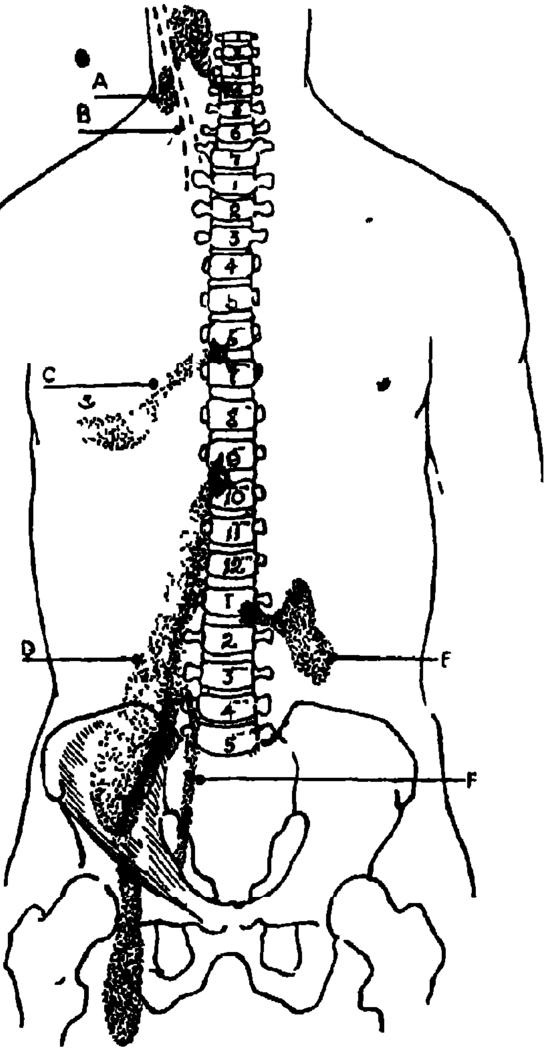

Fig. 125.—Caries of the spine. Diagram of trunk viewed from in front, showing varieties of abscess. A, Retropharyngeal abscess from cervical disease (B, Line of sterno-mastoid muscle); C, Abscess spreading from dorsal caries along the course of one of the intercostal spaces; D, Psoas abscess tracking down from the lower dorsal vertebræ into pelvis and thigh (F, Rare extension of psoas abscess through the sacrosciatic notch into the buttock); E, Lumbar abscess tracking backwards into the loin.

Spondylitis Deformans.—Affects elderly people—Is of very slow development—A large area of the spine is affected—A general kyphotic curve is produced.

Spinal Osteomyelitis.—Symptoms are very acute, with high fever.

Aneurysm of the Aorta.—The pain is intense, constant, and unrelieved by rest. No tenderness exists on pressure on the bones. Other signs of aneurysm may be present.

Malignant Disease of the Spine.—This is usually secondary to some primary focus in the breast or stomach.

Hydatid Disease of the Spine.—This is practically never diagnosed unless other circumstances, e.g., the history of previous hydatid disease, point to it.

Abdominal Disease, especially chronic appendicitis or renal disease, is simulated by the girdle pain in children. In these there are always an absence of spinal signs and a presence of other visceral symptoms.

Disease of the Sacro-iliac or Hip Joints.—In these, local pain, stiffness, or deformities of the affected joints are discovered on examination.

2. CASES OF DEFORMITY IN CHILDREN.—

Rickety Kyphosis.—In this the curve is a general one. Rigidity is but slightly marked. There are no specially tender points over the spine.

3. CASES IN WHICH AN ABSCESS OCCURS WITHOUT PAIN OR DEFORMITY.—These have to be distinguished from the following:—

Tuberculous Abscesses of Other Origin, e.g.—

Hip Disease, or *Sacro-iliac Disease*.—The signs proper to these diseases will be present.

Diseases of the Iliac Bone.—Radiography may indicate the disease, or some local tenderness may occur over the bone.

Lymph-glands (especially in the neck) forming a cold abscess.—Enlarged glands can be felt in the vicinity.

Empyema.—History, signs, and symptoms of pleural and lung disease.

Pyogenic Abscesses, e.g.—

Perinephric Abscess.

Appendicular Abscess.

Abscess from Disease of the Pelvic Organs.

There will almost certainly be signs pointing to the diseased viscus, whilst the spine is mobile and free from tenderness. When these abscesses are opened they smell of the *Bacillus coli*.

In all doubtful cases X-ray photographs in different positions should be taken.

Treatment.—

IMMOBILIZATION.—

Rest in Bed, combined with one of the following.—

Fixation Apparatus for six to twelve months. Plaster jacket or metal frame (*Fig.* 126) for disease of dorsal spine. A stiff collar for cervical disease, or better, a well-fitting plaster-of-Paris jacket of the Minerva or Fillete type.

Immobilization should be continued until all pain and tenderness have disappeared, and a supporting apparatus should be worn for at least six months longer. Average time of treatment is 3 years.

Spinal Fusion Operation.—When the disease is localized to one or two vertebræ in adults, when active disease has ceased. Such an operation is most suitable for adults, and may be of two kinds: (1) ***Albee's bone-graft method***. A graft is taken from the patient's tibia. About five

Pott's Disease—Treatment, *continued.*

spinous processes of the affected vertebral area are exposed and split and the bone-graft is inserted into these (*Fig.* 127). (2) *Hibbs' operation* produces spinal fusion by breaking the spinous processes and laying them down so as to grow together; also by doing an arthrodesis of the intervertebral joints between the laminæ (*Fig.* 128).

ABSCESSES must be treated according to general principles (*see* p. 9).

PARAPLEGIA.—

Rest in Bed with Extension should be applied directly the signs begin; this will arrest the condition in most cases.

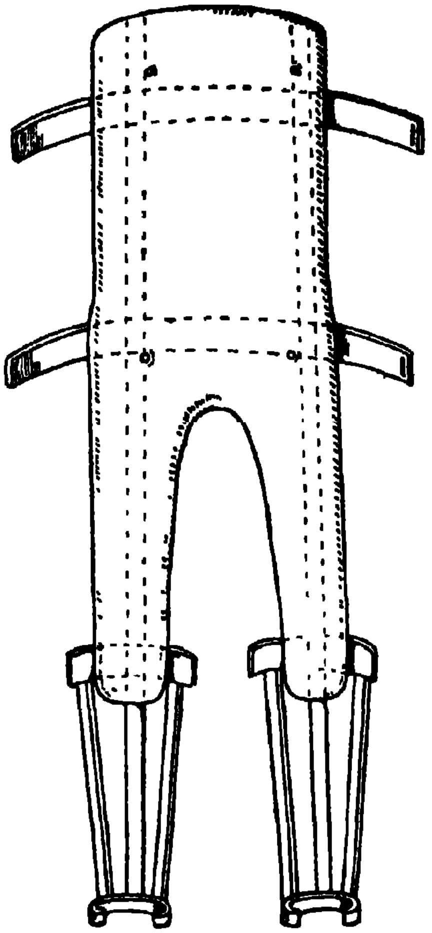

Fig. 126.—Spinal frame.

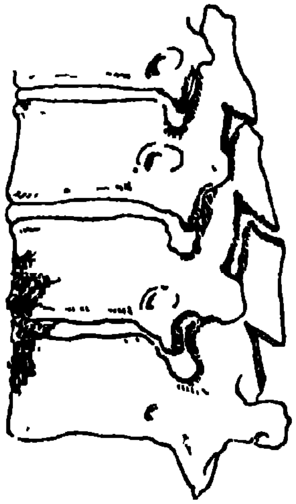

Fig. 128.—Fusion operation for caries of the spine. Hibbs' method. Fracture of spinous processes and arthrodesis of intervertebral joints.

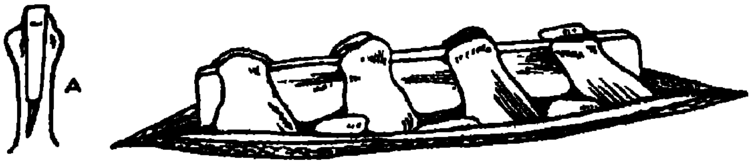

Fig. 127.—Bone-grafting for caries of the spine. Albee's method. Spinous processes are split and the graft laid in the groove so formed. A, Spinous process and graft seen in section.

LAMINECTOMY is done for those cases which do not improve after a month or six weeks of rest and extension.

The laminæ are removed at the site of the angle or over the spot corresponding to the highest position of the paralysed muscles. The dura is pulled to one side without opening, and the abscess sought for at the side and in front of the cord by a bent probe. If found, a soft tuberculous focus may be gently scraped out.

COSTO-TRANSVERSECTOMY.—For abscesses which collect at the side and in front of the vertebral bodies. The neck of the rib and the transverse process are removed and the abscess exposed and scraped out.

SPONDYLITIS DEFORMANS

Definition.—A chronic inflammatory condition characterized by progressive kyphosis, and ending in ankylosis of the spine (poker back), which may be accompanied by acute pain referred along the spinal nerves.

Pathology.—Absorption of intervertebral discs. Synostosis of vertebral bodies. Formation and interlocking of osteophytes, and ossification of spinal ligaments

Varieties.—There are two main types :—

1. VON BECHTEREW'S TYPE.—Affects upper cervical and dorsal regions. Associated with flattening of chest and fixation of ribs.
2. STRÜMPELL-MARIE TYPE or SPONDYLOSE RHIZOMÉLIQUE. —First attacks lower portion of spine and hip, and later affects shoulder.

OSTEOCHONDRITIS OF THE SPINE

Juvenile Type.—Known as Calvé's disease

Occurs in infancy (2–7 years) Affects one vertebra only—usually in dorsal region Causes abnormality in growth and shape of vertebral body, which becomes flattened and more dense Radiograph looks like a penny seen sideways

Discs unaffected. Spontaneous recovery

TREATMENT.—Rest and recumbency

Young Adult Type.—Described by Scheuermann

Occurs as an epiphysitis affecting the surface of the vertebral bodies in young adults Leads to wedging of a number of vertebræ and kyphosis.

ABNORMALITIES OF THE INTERVERTEBRAL DISCS AND LIGAMENTS

Anatomy.—The disc consists of a dense capsule of fibrocartilage, the *annulus fibrosus*, and a semifluid core of gelatinous material, the *nucleus pulposus.*

Herniation into Vertebral Bodies.—In elderly persons the more fluid central part of the disc may be squeezed into the spongy part of the bodies. It accompanies many types of spondylitis and is degenerative in origin.

Herniation into the Spinal Canal.—Usually in the lumbar region.

Probably results from trauma, compression of the spine causing the nucleus pulposus to burst into the spinal canal. Recognized on history and clinical examination, and introduction of air into the spinal theca (air myelography).

Herniation of Intervertebral Discs into Spinal Canal, *continued.*

May cause symptoms of spinal cord pressure. More frequently causes root pressure, low back pain, and sciatica.

TREATMENT.—Removal after laminectomy.

Ligamenta Subflava.—The stout ligaments lining the deep surface of the laminæ. In the lumbar region, after an injury, they may become so thick as to cause pressure on the nerve-roots. Diagnosis and treatment are the same as for the disc lesions.

CHORDOTOMY

An operation for the relief of pain associated with incurable disease in the pelvis, e g., cancer or sarcoma The pain fibres run in the anterolateral tracts of the cord, crossing to the opposite side. It is thus possible to divide one or both anterolateral tracts in the mid-dorsal region through a comparatively small laminectomy.

Resection of Posterior Nerve Roots (Foerster's Operation).—This consists in division of a number of posterior nerve-roots going to the upper or lower limbs or to the intercostal nerves It was suggested for the relief of pain, spasticity, or visceral crises. But the operation had a high mortality and gave very uncertain results, so that it has been superseded by the operations of chordotomy and those on the sympathetic nerves (*see* Chapter XVII).

CHAPTER XXV

HEAD INJURIES

INJURIES OF THE SCALP

Hæmatoma of the Scalp may follow injury. If infected the hæmatoma may form an abscess of the scalp

1. SUPERFICIAL TO APONEUROSIS.—Small and ill-defined. Trivial.
2. SUB-APONEUROTIC.—Large and ill-defined. Only limited by attachments of occipito-frontalis. If septic, may be fatal
3. SUB-PERICRANIAL (CEPHALHÆMATOMA) Limited to one or other skull bone owing to pericranium dipping between sutures. Commonly seen in birth injuries May be associated with underlying fracture Closely resembles depressed fracture of skull

TREATMENT —Leave hæmatoma to subside If infection occurs, drain as necessary.

Wounds of the Scalp may be incised, lacerated, or punctured.

Three features require special note:—

HÆMORRHAGE is very free, either from partial division of an artery or from the difficulty of securing a wounded vessel in the dense tissues of the scalp. It is often necessary to pass a suture through whole thickness of scalp to stop bleeding

SEPSIS is frequent from the hair and hair follicles, and readily leads to cellulitis

The neighbourhood of all wounds should therefore be freely shaved.

AVULSION of part or the whole of the scalp may occur, especially in women,

from entanglement of hair in machinery

If the separated part can be found, and is fairly clean, it should be replaced and sutured, and has then a fair chance of re-union. Otherwise extensive skin-grafting will be required

Cellulitis of the Scalp is of serious import, for the following reasons :—

1. EXTENT —It rapidly spreads beneath the aponeurosis so as to extend from the forehead to the occiput.
2. MENINGEAL INFECTION readily occurs by extension through the veins and lymphatics through the skull.
3. CRANIAL OSTEOMYELITIS may result by infection of the veins of the diploë.

TREATMENT must be by early multiple incisions in the most dependent situations, free drainage, and repeated fomentations, and chemotherapy.

Traumatic Cephalhydrocele.—Occurs in children, but is very rare. Fluctuating fluid swelling under the scalp. Communicates with subarachnoid space or lateral ventricle through a fracture in the vault. Pulsates with the heart and respiration.

FRACTURES OF THE SKULL

Fractures of the skull are of great importance when they are:—

1. Open—liability to infection of brain and meninges.
2. Depressed—liability to compression of brain and meninges.
3. Associated with hæmorrhage—liability to compression of brain and meninges.

Occasionally fractures are of localizing value. Otherwise they are of no significance in themselves; it is the associated degree of cerebral damage that matters.

Mechanical Factors.—The skull being an elastic sphere is capable of some yielding to a blow This involves a bending in at the point of contact and at the opposite pole, and a bulging out at the equator between these poles. Thus, all cranial fractures may be divided into:—

1. BENDING FRACTURES.—Occurring at the point of impact and at that opposite to it, from compression at the poles. Commoner at the vault. THE INNER TABLE suffers more than the outer, and over a wider area. It may be the only part fractured. In infants and the aged, in whom no diploë exists, the vault only breaks in one piece.
2. BURSTING FRACTURES—Caused primarily by expansion between the poles of impact and resistance. Occur as radiating fissures. Commoner at the base, where thin areas and foramina lie between thick resistant areas. The fissures run most commonly down the temporal fossa of the vault into the middle fossa of the base and across the sella turcica This area lies between the strong buttresses formed by the external angular process of the frontal and lesser wing of the sphenoid in front and the mastoid and petrous bones behind.
3. PUNCTURED FRACTURES.—These may be considered here under the commonest variety, viz:—

Gunshot Fractures.—The path of the bullet may be directly perforating or tangential

IN PERFORATING FRACTURES, the table last traversed is most injured; thus, at the wound of entry the inner table is most broken, and at the wound of exit the outer

THE EXPLOSIVE EFFECT of the hydrodynamic force transmitted to the fluid brain by the bullet causes a bursting fracture with much comminution.

EFFECT OF VARYING FORCE.—A modern rifle at close range causes extensive comminution; at about a mile, simple perforation; a rifle beyond a mile, or a pistol, may not penetrate, or the bullet may lodge.

TREATMENT should be on general lines, according to brain symptoms. Attempts to probe for and remove bullet should never be made unless there are definite symptoms of local irritation.

General Considerations.—

RELATION BETWEEN BASE AND VAULT FRACTURES.—The majority of fractured vaults have also a fractured base. Basal fractures often occur alone.

IMMEDIATE MORTALITY.—About one-third die within forty-eight hours from cerebral complication

LATE MORTALITY.—Immediate survivors often die of meningitis.

REPAIR OF CRANIAL FRACTURES is slow, incomplete, or fibrous, but if there is no cerebral damage this fact is of no consequence.

FRACTURES OF THE VAULT

Varieties.—Fissured (no symptoms or signs of fracture unless open)—Depressed—Punctured. (*See also Fig.* 129.)

Depressed and Punctured Fractures.—Generally comminuted. In children depression may exist without fracture. Outer or inner table may alone be broken.

Inner table is generally much more broken than outer because it is less supported; it is broken by a force of less momentum; the breaking force is radiated over a wider area.

VARIETIES.—

Closed (Simple) or Open (Compound)

Pond.—No sharp depressed edge

Gutter.—With sharp depressed edge.

Gunshot.—Wound of entry has inner table most damaged. Wound of exit has outer table most damaged. May be associated with fractured base.

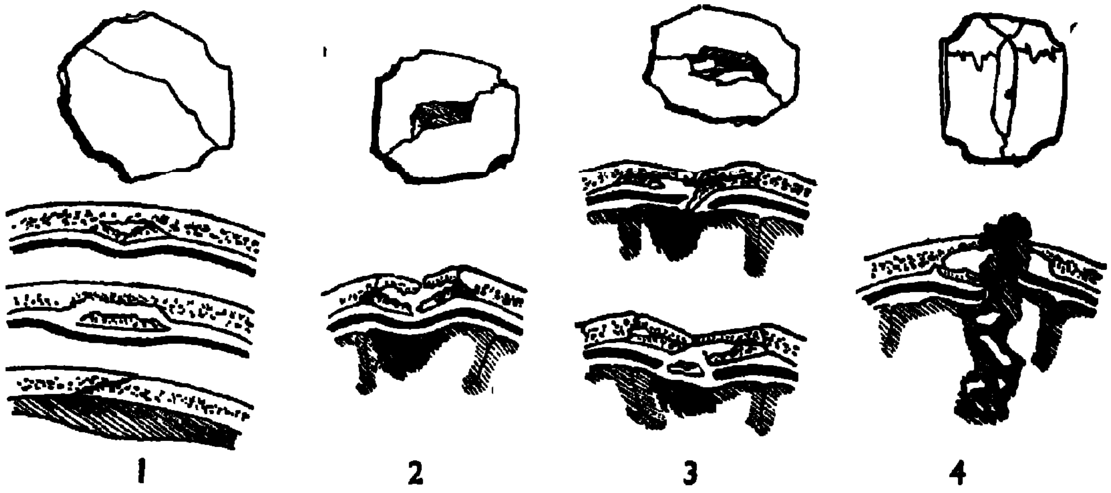

Fig. 129.—Fractures of the cranial vault (*after Cushing*). 1, Simple fracture, with splintering of the inner table, 2, Fracture of both tables, with contusion of the brain; 3, Fracture of both tables, with laceration of the dura; 4, Fracture of skull, laceration of dura, with particles of bone driven into the brain.

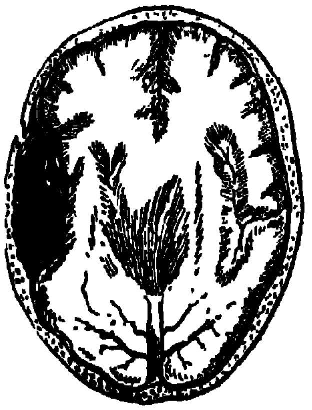

Fig. 130.—Head shown in horizontal section. There is a fracture of the skull, with a subcranial or extradural hæmorrhage between the bone and dura, from a laceration of the middle meningeal vessels. Note the compression of the brain.

Fractures of Vault of Skull, ***continued.***

Symptoms.—

1. Concussion from the blow.
2. Compression from pressure of bone or exudate.
3. May be No Cerebral Symptoms at all.
4. If Open and Septic: Symptoms of septic infection—Necrosis of bones or osteomyelitis, pyæmia—Epidural or subdural abscess—Meningitis—Cerebral abscess—Late cerebral compression—Possibly hernia cerebri.

Treatment.—

The general treatment and the operative treatment for signs of compression are listed in the section on fractured base (*see* p. 295).

Wounds associated with compound fractures must be carefully cleaned, with free scalp shaving.

In Comminuted and Punctured Fractures, remove loose spicules of bone, especially those of the inner table. Remove subdural clots if these are present

In all Depressed Fractures trephine beyond the fracture and elevate. Remove loose bone from the inner table.

Birth Fractures.—Depressed or fissured.

DEPRESSED —Due to forceps Signs and symptoms vary according to depth of depression The fracture may be concealed by cephalhæmatoma

Treatment.—Elevation. If symptoms present, operation is urgent; otherwise elevation at leisure.

FISSURED —Due to falls or blows Differ from those of adult in that the width of the fissure tends to increase owing to increasing intracranial pressure from growth of brain, and traumatic cephalocele may result.

FRACTURES OF THE BASE

Causes.—

INDIRECT VIOLENCE, acting on the vertex —By radiation of fracture from the vertex By compression of the elastic skull, causing it to burst at the weak points at the base

DIRECT VIOLENCE —Penetrating wounds through orbit or nose Blows transmitted through condyles of jaw Violence transmitted through the vertebral column.

Position of Fractures.—Generally transverse through the fossæ. Often run right across the base Often run from one middle fossa to the opposite anterior or posterior fossa. Generally traverse the nerve foramina. Petrous bone is often involved

Varieties.—

CLOSED.—Especially in the posterior fossa.

OPEN.—The commonest variety.

In Anterior Fossa.—Into nose or orbit.

In Middle Fossa.—Into external or middle ear; into nasopharynx; into sphenoidal sinuses

In Posterior Fossa.—Into nasopharynx.

Brain may be concussed, contused, lacerated, or compressed.

COMPLICATED.—

MENINGES may be lacerated. Meningitis, simple or septic, may follow.

VESSELS may be contused, with resulting thrombosis, or lacerated.

Venous sinuses—subdural hæmorrhage.

Middle meningeal artery—extradural hæmorrhage (*Fig.* 130).

Internal carotid artery—aneurysm or fatal hæmorrhage.

NERVES may be damaged or torn: Sensory loss—Paralysis.

Signs of Fractured Base.—

USUAL SIGNS are those of the complications.—

1. CEREBRAL CONCUSSION OR LACERATION.—Usually prolonged coma.
2. HÆMORRHAGE.—

   Bleeding from the Nose—From fracture of ethmoid (anterior fossa): basi-sphenoid (middle fossa); basi-occipital (posterior fossa). May be swallowed and then vomited

   Orbital Hæmorrhage (anterior fossa)—Ecchymosis (chiefly of the lower lid). Subconjunctival (below the cornea without posterior limit). Proptosis. Orbital aneurysm—internal carotid artery and cavernous sinus both injured.

   From Ear (middle or posterior fossa).—Laceration of small vessels in middle ear, together with rupture of drum. Injury of internal carotid or one of the venous sinuses Injury of drum, cartilage, or meatus may cause slight bleeding independently of fractured base.

   Into Muscles at Back of Neck (posterior fossa).—Shown by ecchymosis, which may not be apparent for days
3. DISCHARGE OF CEREBROSPINAL FLUID—From the nose, or more commonly the ear Caused by laceration of dura, generally in the internal meatus. Large quantity (one or more pints).

   Fluid with the following characters· Sp. gr. 1005—Alkaline—Does not coagulate on boiling—Reduces copper.
4. ESCAPE OF BRAIN SUBSTANCE FROM THE NOSE OR EAR.—Rare and only in fatal cases
5. NERVE LESIONS—

   Optic nerve or tract· rare Blindness.

   Third, fourth, sixth nerves common Ocular paralysis

   Seventh and eighth: the commonest of all. Facial paralysis and deafness.

   Ninth, tenth, eleventh, and twelfth: rarely involved.

   Lesion may be immediate, from rupture, often permanent; or later from callus involvement, generally recovers.

Prognosis of recovery is that of the complications. The majority of deaths occur within forty-eight hours of injury. If marked improvement does not occur before this time the prognosis is grave. Thus, in uncomplicated cases it is good; in cerebral lesions, in rupture of large vessels, and in septic complications (meningitis or abscess) it is bad.

Prognosis of Completeness of Recovery is seldom good, because of the sequelæ.

Treatment.—(N.B.—The treatment is for the complications. The fracture itself requires no special measures, apart from steps to prevent infection.) Disinfection and dressing of the external auditory meatus. Shave the head. Apply cold by ice-bags or Leiter's tubes. Keep at absolute physical and mental rest for six weeks. No mental work for three to six months. Lumbar puncture both in diagnosis and treatment. Dehydration therapy, by mouth.

Fractures of Base of Skull—Treatment, ***continued.***

OPERATIVE TREATMENT.—If signs of compression are well marked or increasing during the second day, a subtemporal removal of bone should be performed, if necessary on both sides. The dura is opened and the subdural space drained. (*See* CEREBRAL COMPRESSION, p. 297.)

INTRACRANIAL COMPLICATIONS OF HEAD INJURIES, ETC.

Varieties.—

CEREBRAL.—(1) Concussion; (2) Irritation; (3) Contusion; (4) Compression; (5) Laceration.

INFLAMMATORY.—(1) Subcranial abscess; (2) Meningitis; (3) Sinus thrombosis; (4) Cerebral abscess

HÆMORRHAGE.—(1) Venous sinuses; (2) Extradural vessels; (3) Subdural; (4) Subarachnoid; (5) Cerebral.

(The inflammatory complications are described in Chapter XXVI.)

CEREBRAL LESIONS

Cerebral Concussion.—

PATHOLOGY —Engorgement of viscera. Ecchymosis of brain, but no other macroscopic change

Theories as to cause: (1) Molecular disturbance of brain cells; (2) Multiple minute contusions, (3) Paralysis of vasomotor centre produced by a sudden acute cortical anæmia. The last is probably correct.

SYMPTOMS —

UNCONSCIOUSNESS, partial or complete.

PUPILS equal, moderately dilated and sluggish in reaction. In fatal cases they are equal, dilated, and fixed.

SURFACES pale, cold, clammy.

RESPIRATION slow, shallow, irregular.

TEMPERATURE subnormal

PULSE weak, rapid, and irregular.

REFLEXES present in all but worst cases

BLADDER AND RECTUM unaffected except in worst cases.

FOLLOWED BY one of the following conditions:—

1. REACTION —Return of consciousness Rise of temperature to 101°–102°. Headache and vertigo. Vomiting. Some irritability. Later for some days there may be a subnormal temperature and slow pulse.
2. DEATH.—Deepening unconsciousness. Temperature rises to 104° or over. Fatal cases of concussion are extremely rare Autopsy usually reveals a definite lesion of the brain.
3. CEREBRAL IRRITATION, INFLAMMATION, OR COMPRESSION.

TREATMENT.—Rest in bed for several weeks. Ice-cap to the head. Light diet.

Cerebral Irritation.—This is a clinical rather than a pathological conception. It is associated with certain cases of concussion, especially with those where contusions of the frontal and occipital regions have occurred.

SYMPTOMS.—The symptoms of concussion gradually give place to the following Great mental irritability whenever aroused from drowsy stupor. Headache. The patient lies curled up in a position of general flexion. The drowsiness of concussion gives place to restlessness or delirium. The pulse and respiration are irregular. The irritability is succeeded by fatuity. May last from 48 hours to 14 days.

TREATMENT.—As for concussion, but these cases especially require prolonged mental and physical rest, in order to prevent permanent mental or epileptic symptoms Lumbar puncture is helpful and the cerebrospinal fluid pressure is reduced gradually. Magnesium sulphate solution by the mouth.

Cerebral Contusion.—This is practically a severe grade of concussion or a slight grade of laceration.

THE USUAL SITES are at the tip of the temporal lobes and the under side of the frontal lobes, often produced on the side opposite to the blow by *contre-coup*.

SYMPTOMS.—Those of concussion, but more prolonged in duration, and nearly always succeeded by some degree of compression or irritation. Convalescence is prolonged and sequelæ are more marked than with simple concussion.

LUMBAR PUNCTURE shows blood in the cerebrospinal fluid.

TREATMENT—Rest in bed, until headache, slow pulse, and retinal stasis have disappeared Cases that do not rapidly improve, and where repeated lumbar puncture shows persistent rise of pressure, should have exploratory trephinement to exclude subdural hæmatomata.

Cerebral Compression.—

CAUSES.—Depressed fractures—New growth of bone—Blood (extradural, subdural, intracerebral)—Œdema—Pus (extradural, subdural, cerebral abscess)—Tumours.

PHYSIOLOGY—There are two types of compression of the brain.—

1. LOCAL, e g., that caused by a tumour
2. GENERAL, e g., that of hydrocephalus, in which the whole intracranial circulation is affected

CIRCULATORY CHANGES.—The cerebrospinal fluid is first driven out. Then the blood-vessels are compressed, the veins suffering first because of their thin walls and low blood-pressure Venous stasis is succeeded by capillary anæmia This causes loss of function in the anæmic parts, and death if these be the vital centres in the medulla.

DIVISION OF THE CRANIAL CAVITY—The cranial cavity is divided by the falx and tentorium into three spaces, and thus a local compression may reach a high degree before causing general compression. Subtentorial compression is the most important, because the condition of the medulla is the crux of the situation.

COMPENSATORY MECHANISM.—If subtentorial pressure exceeds that in the arteries, death must result from anæmia of the medulla. But the early stages of stasis or slight anæmia stimulate the vasomotor centre, and the blood-pressure is raised This goes on until the blood-pressure may be twice as high as normal. At last the blood-pressure can rise no longer, intracranial pressure is the higher, and death results.

FLUCTUATIONS IN BLOOD-PRESSURE—There is a regular oscillation in blood-pressure as the pressure in the cranium and in the vessels of the vasomotor centres are balancing one another. Hence the alternations of blood-pressure and of respiratory rhythm, which correspond to these changes in the medullary circulation. This explains Cheyne-Stokes respiration.

Cerebral Compression, ***continued.***

SYMPTOMS.—

1ST STAGE—Compensation. Symptoms are few. Headache, with mental dullness.

2ND STAGE—Venous stasis. Severe headache, with drowsiness or restlessness. Congestion of the vessels on the scalp and eyelids. The fundus oculi shows swelling and enlarged tortuous veins. The pulse is slow and the blood-pressure rises.

3RD STAGE—Cerebral anæmia. Unconsciousness absolute.

Respiration slow, laboured, and stertorous Cheeks puffed out. Soft palate paralysed. Cheyne-Stokes breathing in bad cases.

Pulse full, slow, Blood-pressure is high.

Temperature generally raised. More on the paralysed side than the other.

Pupils unequal and fixed. Each first contracts, then dilates; first the one on side of lesion, then the opposite. Choked disc.

Motor Paralysis, generally hemiplegic.

Bladder paralysed, retention.

Rectum.—Sphincter paralysed.

Reflexes Differ on two sides generally. On paralysed side they are increased

4TH STAGE—Medullary paralysis.

The respiration fails first, and then the pulse becomes rapid and weak. The pupils are widely dilated and fixed The blood-pressure falls and death occurs.

TREATMENT.—

GENERAL TREATMENT as for concussion

LOCAL COMPRESSION.—Remove the cause if possible, e.g , by elevating fractures, removing blood-clots or tumours, opening abscesses, etc.

INTRAVENOUS INJECTION TO REDUCE INTRACRANIAL TENSION.—Intravenous 50 per cent sucrose is very satisfactory. Intravenous hypertonic saline only brings about a temporary improvement and may be followed by a more severe reaction.

VENESECTION is a time-honoured remedy of great danger. It lowers the blood-pressure, and so weakens the compensatory mechanism which prevents the medulla from becoming anæmic

LUMBAR PUNCTURE controlled by manometry is useful unless a posterior fossa tumour is suspected, when lumbar puncture is highly dangerous.

SUBTEMPORAL DECOMPRESSION.—This should be carried out in all cases where a local cause cannot be removed, and in which the symptoms are those of the third stage. The temporal muscle is split, a piece of bone about 1½ in. in diameter is removed towards the base of the skull, the dura is opened, and a drain inserted It should be done on both sides unless a definite cause is found on the first side explored Post-operative treatment: rest, darkened quiet room, and adequate time in bed, i.e., 28 days, followed by a prolonged convalescence.

Cerebral Laceration.—

CAUSES.—

1. Blows on the head. Common complication of fractures of the skull. Occurs either: (*a*) Beneath point struck, or (*b*) A point opposite from *contre-coup*.
2. PENETRATING WOUNDS, with compound fracture.

ANATOMY.—

IMMEDIATE.—Laceration or pulping of nerve substance—Pial hæmorrhage —Ecchymosis in brain substance—Bleeding may be into the ventricles.

LATER.—Local œdema—Spreading œdema, with increase of the cerebrospinal fluid—Red softening—Yellow softening, or rarely an arachnoid cyst.

SYMPTOMS.—There are none which are really peculiar to laceration, but the following conditions are strongly suggestive of it:—

1. Symptoms of concussion, merging into those of compression.
2. Symptoms of concussion, with coma which does not lessen.
3. Symptoms of concussion, followed by those of irritation.
4. Convulsions affecting regular groups of muscles, leaving convulsed parts paralysed, and spreading in a regular sequence.
5. Temperature rising to 104° F. or over is suggestive of laceration of a severe kind.

INTRACRANIAL HÆMORRHAGE

VARIETIES —(1) Extradural—between the dura and cranium; (2) Subdural—between the dura and arachnoid; (3) Subarachnoid—between the arachnoid and pia; (4) Cerebral—in the substance of the brain.

Extradural or Subcranial Hæmorrhage.—

CAUSES.—Ruptured middle meningeal artery in the great majority of cases. Rupture or wounds of the venous sinuses or other meningeal vessels. Rarely in the frontal and occipital regions. Extradural hæmorrhage is very rare in infants or aged people, because in both these the dura is so closely attached to the skull.

MECHANISM.—The middle meningeal artery runs in a deep groove or tunnel in the sphenoid, temporal, and parietal bones, and therefore is often torn in fractures of the base or vertex, or by a blow which separates the dura without fracturing the skull. May be on the side opposite to the blow. Usually the anterior branch which suffers The blood collects between the dura and the skull, driving the former in upon the brain (*see Fig* 130, p 293).

SYMPTOMS.—Typically there are three stages:—

1. Concussion from the blow, with signs and symptoms of fractured skull; blood-pressure is low and hæmorrhage is slight.
2. Recovery from unconsciousness, with improving pulse. The blood-pressure rises, and bleeding strips the dura from the bone.
3. Gradual coma from cerebral compression by the blood-clot This comes on within a few hours or days of the return to consciousness. Retinal congestion with choking of the disc is one of the earliest signs, and occurs first and in most marked degree on the side of the lesion.

And/or in bad cases the signs of concussion may merge into those of compression without a conscious interval.

Convulsions and paralysis of the arm and face on the opposite side may occur as the coma sets in. Aphasia may occur in left-sided injuries.

Congestion of the eye of the same side, with proptosis and ocular paralysis, may result from pressure on the cavernous sinus.

Swelling and ecchymosis of the temporal fossa occur from co-existing injury or from leaking of blood through a fracture. Typical pupil changes as described by Hutchinson (*see* CEREBRAL COMPRESSION).

Extradural Hæmorrhage, *continued.*

DIAGNOSIS.—Is only possible when the typical sequence of symptoms occurs.

From INTRAMENINGEAL HÆMORRHAGE by the delayed onset of coma.

From CEREBRAL LACERATION by the absence of marked rise of temperature and by the 'lucid interval'.

Also a lumbar puncture withdraws clear fluid containing no blood-corpuscles.

TREATMENT.—Trephine and clear out the blood-clot, plugging or tying the meningeal artery; this must be attempted as soon as the diagnosis is made. In doubtful cases it is safer to explore Explore the opposite side if nil found on the suspected side. The posterior branch of the artery may also need exploration.

PROGNOSIS is bad, because the operation is usually delayed too long, so that the compressed brain cannot recover

Intrameningeal Hæmorrhage (including subdural and subarachnoid bleeding).—

CAUSES.—Injuries of the meningeal vessels, especially those of the pia mater, also of the venous sinuses, by punctured wounds or fractures of the skull In people over 40 years minor degrees of injury may cause a chronic type of subdural hæmorrhage and a subdural hæmorrhagic cyst may form.

ANATOMY —The blood is spread out between the brain and the dura, compressing the former. If the case recovers, the blood is absorbed and an ARACHNOID CYST left.

SYMPTOMS are those of cerebral compression coming on directly after an injury. Convulsions and paralysis occur if the motor area of the cortex is affected Late paralysis or Jacksonian epilepsy may occur from the presence of adhesions or a cyst

DIAGNOSIS is almost impossible from severe concussion and laceration of the brain.

Spinal fluid from a lumbar puncture contains blood-cells.

TREATMENT —All cases in which coma lasts, and intracranial pressure is high, should be trephined in the subtemporal region on both sides if necessary. If the dura is found to be blue and bulging, it is opened, the blood and clot are removed, and a drain is inserted.

Cerebral Hæmorrhage.—The chief importance of this is its recognition as differing from other forms of bleeding which are amenable to surgical measures. Usually it occurs apart from trauma in cases of Bright's disease or high arterial tension. Localized focal signs of hemiplegia are more marked than the other signs of compression.

Intracranial Hæmorrhage in the New-born.—

CAUSES.—The moulding of the soft cranial bones during difficult labour. The pressure of obstetric forceps.

ANATOMY.—The over-riding of the edges of the parietal bones tears the veins which enter into the superior longitudinal sinus. The blood is poured out over the surface of the brain in the region of the median longtitudinal fissnre or over one or both hemispheres.

SYMPTOMS.—Asphyxia or delayed and irregular respiration. The anterior fontanelle is tense, bulging, and pulsates feebly. The scalp and eyelid vessels are dilated. The pupils are unequal. Convulsions are common and often fatal. Paralysis of cortical origin is not seen in infants.

RESULTS.—The majority die within a few days of birth from convulsions or respiratory failure. In the survivors, various birth palsies (Little's disease) develop in later life, with dementia or idiocy. Bilateral spastic paralysis of the legs is common from involvement of both leg centres near the longitudinal fissure. Spastic diplegia occurs when both arm and leg centres on the same side are involved.

TREATMENT.—Repeated lumbar punctures to control pressure. In special cases exploration of the dura and cortex through an enlarged burr hole.

REMOTE RESULTS OF HEAD INJURIES

PERCENTAGE.—About half of all cases of fractured skull and concussion show some slight permanent sequelæ. Ten to twenty per cent have a marked after-effect.

DISPOSITION to mental after-effects is determined by: (1) Natural temperament, (2) Alcoholic habits; (3) Youth or old age; (4) Efficiency of treatment—operation, and long rest afterwards, lessening the chances of after-effects.

TREATMENT.—In general the established lesion is difficult to cure. Prevention by effective primary treatment is the ideal. Provided some local focus can be found, operation is beneficial—in many cases no such focus exists. Until exploration has been carried out the possibility of a focus cannot be excluded.

Remote Symptoms.—

1. General Cephalalgia.—Constant dull pain over frontal, vertical, or occipital regions.

2. Local Cephalalgia.—Headache with local tenderness May be due either to osteosclerosis (when it is cured by trephining), or meningeal adhesions

Painful Scars.—Caused by the adhesion of the scalp to the cranium.

Vertigo.—Is left in 22 per cent of fractured skulls, and is due to cerebral instability.

Vomiting is often associated with the vertigo.

Mental Changes.—(*a*) Melancholia, suicide (1–2 per cent); (*b*) Irritability; (*c*) Loss of 'nerve'; (*d*) Inability for mental exertion; (*e*) Disturbed sleep; (*f*) Liability to alcoholism and sunstroke.

. Motor Aphasia only follows injuries to Broca's centre.

. Amnesia, or loss of memory, occurs very commonly (35 per cent) in a slight degree. It results from mental instability and failure of power of attention. It may be one of three types: (*a*) Loss of memory of the accident and the following days: this is usual. (*b*) Loss of memory of the life previous to the injury: this is rare for long periods, but common for short periods—medico-legal importance. (*c*) Loss of memory for events subsequent to the injury: this is common.

. Special Nerve Injuries.—Loss of smell, inequality of pupils, accommodative asthenopia, nystagmus, optic atrophy, deafness (12 per cent), increased knee-jerks, slow pulse.

Results of Head Injuries—Remote Symptoms, ***continued.***

10. GLYCOSURIA.—Temporary is common. Permanent very rare. More frequently follows injuries of the back of the head.

11. HEMIPLEGIA.—Primary, or secondary from scar, cyst formation, or sclerosis.

12. TRAUMATIC EPILEPSY.—Occurs in 7 per cent of fracture cases. The majority are idiopathic or general epilepsy, but one-third are of a Jacksonian type.

In Jacksonian or traumatic epilepsy, the convulsions precede loss of consciousness, and the latter may be slight or absent. The convulsions usually begin in the same group of muscles, i.e., in those related to the affected cortical centre From this group the convulsions spread to neighbouring groups until, in bad cases, they may become general. The muscles first convulsed remain paralysed for some time after each convulsion.

Prophylaxis..—If all depressed fractures and punctured cranial wounds were operated on, it would seldom occur.

Treatment.—Operations are usually very unsatisfactory. For the general type they are useless, and in Jacksonian epilepsy the symptoms often recur.*

Cases suitable for operation are those with: (*a*) A definite injury and scar; (*b*) Localized convulsions, (*c*) History of less than two years; (*d*) Absence of family history of epilepsy or insanity.

Operation.—After exposing the brain and freeing it from the dura, remove clots, cysts, or massive scars. If no gross exciting cause can be found, the cortical area connected with the muscles which initiate each convulsion should be excised in suitable cases. Place gold-leaf between the brain and the dura.

13. TRAUMATIC INSANITY.—Occurs in about 7 per cent. In the majority the injury merely precipitates a condition to which the patient was disposed. In cases where the injury leads directly to insanity, the lesion is generally over the left pre-frontal or parietal regions.

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* Cushing, however, gives the following figures: Of 128 cases submitted for treatment, only 59 were operated on. Of these 59, 12 were cured (for periods of 1 to 5 years), 30 were improved, and 17 remained unaffected, of whom 2 died in a status epilepticus. (Keen's *System of Surgery*, vol. iii, p. 251.)

CHAPTER XXVI

DISEASES OF THE CRANIUM, BRAIN, AND MIDDLE EAR

DISEASES OF THE SCALP

Simple Tumours.—

PAPILLOMA.—Commonly multiple; may require excision.

FIBROMA.—As a huge pendulous mass, or else a moderate-sized tumour. TREAT by excision.

SEBACEOUS CYSTS are very common. Move with the scalp freely over the cranium, and there is no depression in the bone. They may suppurate or fungate, when they form a foul exuberant mass.

TREAT by excision.

DERMOID CYSTS occur near the outer canthus of the eye. They are usually attached to the skull, or buried in a cavity in its surface, or attached to the meninges by a pedicle which goes through the skull.

TREAT by excision, for which the skull may have to be opened.

LIPOMA is lobulated and generally under the aponeurosis, and the scalp moves over it. Usually occurs in the frontal region.

NÆVUS.—*See* PULSATING TUMOURS, below.

PNEUMATOCELE is an air-containing tumour over the frontal, temporal, or occipital regions. It arises by a communication between the frontal or mastoid air-cells and the cellular tissue of the scalp. Caused by injury or violent sneezing.

TREAT by incision and packing of bone cavity after enlargement of the opening.

Malignant Tumours.—

EPITHELIOMA.—Usually begins at or near the ear. It may, however, start as a papilloma or sebaceous cyst. It has the usual characters, including the implication of lymph-glands.

TREATMENT on usual lines.

RODENT ULCER.—Usually starts from the face and spreads upwards.

SARCOMA.—Is rare as a primary scalp tumour.

Pulsating Tumours.—

NÆVUS.—Especially over the fontanelle, from which it may derive pulsation.

TRAUMATIC ANEURYSM — ARTERIOVENOUS ANEURYSM — ARTERIAL VARIX (temporal)—CIRSOID ANEURYSM.—Most of these occur more commonly in the scalp than elsewhere (*see* pp. 125, 126, and 132).

Traumatic Swellings of the Scalp.—Hæmatoma—Cephalhydrocele (*see* p. 291).

Inflammatory Swellings of the Scalp.—Abscess, subpericranial, subaponeurotic, or superficial (*see* p. 291).

DISEASES OF THE CRANIAL BONES

Simple Tumours.—

OSTEOMA.—Cancellous or ivory. Cancellous generally found in relation to frontal sinus or external auditory meatus; may occlude the latter. Ivory generally found in frontoparietal region. They are of slow painless growth, have a broad base, and abrupt edge. Occasionally occur in accessory nasal sinuses.

TREATMENT.—Removal by chisel and saw. Ivory type is very hard. and it is necessary to remove surrounding bone.

LEONTIASIS OSSEA.—*See* p 328.

HYPEROSTOSIS —Certain meningiomas are associated with hyperostosis of the skull, just as others cause osteoporosis. Most of the old 'sarcomas of the skull' were meningiomatous hyperostoses

Malignant Tumours.—

SARCOMA —May be primary or secondary. A history of injury commonly precedes the disease Begins as a periosteal spindle-cell sarcoma. All are practically inoperable, except possibly the myeloid growth.

CARCINOMA is always secondary, often to carcinoma of breast, thyroid, or prostate.

Pulsating Tumours of the Skull.—Any of the above forms of sarcoma or carcinoma may pulsate.

Aneurysm by anastomosis may occur in the cranial diploe.

Acute Inflammatory Swellings.—

PERIOSTITIS or OSTEOMYELITIS may complicate any wound, fracture, or contusion Will lead to (1) Extracranial abscess, (2) Subcranial abscess, (3) Necrosis of the skull ; (4) Pyæmia or septicæmia.

TREATMENT is by free opening.

Chronic Inflammatory Swellings.—

SIMPLE PERIOSTITIS —From carrying weights on the head or after a blow.

SYPHILITIC PERIOSTITIS.—Hard and soft nodes, the latter being the commoner (*see* p 222).

TUBERCULOUS DISEASE —*See* p. 220

PARROT'S NODES over the frontal and parietal eminences occur in infants with rickets and congenital syphilis There are other evidences of these diseases.

HYDROCEPHALUS

PHYSIOLOGY —The cerebrospinal fluid arises as a secretion or exudation of the ependyma of the choroid plexuses in the ventricles. It escapes from the fourth ventricle through the foramina of Magendie and Luschka into the subarachnoid space. It drains off into the veins, especially into those which enter into the superior longitudinal sinus. Hydrocephalus is caused by an abnormal accumulation of fluid in the ventricles (closed hydrocephalus) or in the ventricles and over the surface of the brain (sommunicating hydrocephalus).

VARIETIES.—Hydrocephalus may be produced by: (1) Excess of cerebrospinal fluid; (2) Interference with the circulation of the fluid; (3) Interference with absorption. Compensatory hydrocephalus follows cortical atrophy.

1. Excess of Cerebrospinal Fluid.—An excess production results from a chronic or acute congestion of the vessels involved in the production of cerebrospinal fluid, e g., the acute hydrocephalus which may accompany tuberculous meningitis. Engorgement of the choroid plexus of the lateral ventricle resulting from thrombosis of the efferent veins of Galen gives an excess production.
2. Interference with Circulation of Cerebrospinal Fluid.—Is either (*a*) Ventricular, or (*b*) Extraventricular.
   - *a*. *Ventricular.*—Caused by adhesions in or over the roof of the 4th ventricle from a birth hæmorrhage, transient mild meningitis in infants, syphilitic meningitis. It may also be due to pressure of a tumour on the foramen of Monro, the iter, or the 4th ventricle.
   - *b*. *Extraventricular* —Caused by inflammatory changes over the cerebral convolutions, and blocking of the apertures at the tentorium cerebelli by inflammatory exudates, tumours, and hæmorrhage.
3. Interference with Absorption of Cerebrospinal Fuid.—Absorption takes place through the arachnoid villi which project into the venous sinuses. Chronic inflammatory changes in relation to these will seriously affect absorption and discharge of the cerebrospinal fluid into the venous blood-stream.

DIAGNOSIS.—Inquire for syphilis, birth injuries, transient meningitis, and such symptoms as may point to the presence of tumour.

Signs.—The cranial cavity is increased, but the bones are much thinned, and in infancy the fontanelles and sutures gape.

Aetiology —Usually in a child, sometimes associated with maternal hydramnios. Several members of the family may have it. In some cases there is a relation to syphilis The ventricles of the brain are greatly distended, and the cerebral substance is atrophied. In extreme cases fluctuation and bony crackling may be felt. The forehead becomes protuberant, and the eyes are pushed forwards. The face is disproportionately small, the ears and eyes being overhung by the bulging cranium. The veins of the scalp and eyelids are engorged.

Symptoms.—Child can hardly raise its head. It hardly ever cries, because crying increases the intracranial pressure. Vomiting is common, and there is great wasting Blindness Usually succumbs to an intercurrent disease. If survival occurs, it is associated with mental deficiency and physical weakness.

Aids to Diagnosis as to Type.—

1. *Phenolsulphonephthalein Test* —Inject phenolsulphonephthalein into lateral ventricle. Later, lumbar puncture, and if by addition of an alkali the phthalein reaction is produced, then the communication between intra- and extra-ventricular streams is complete—i.e., hydrocephalus is communicating.
2. *Ventriculography* —Pass a needle through the anterior fontanelle into the lateral ventricle, and after removal of some cerebrospinal fluid inject its equivalent of air or oxygen. The head is then screened or a series of X-ray photographs taken. The position of the head is changed so as to pass the gas from ventricle to ventricle.

TREATMENT Tapping the ventricles is useless. The only hope is to limit the formation of cerebrospinal fluid from the ventricles of the brain. Dandy and others have perfected a technique and now obtain good results by using a ventriculoscope. The operation is performed through a small burr hole just to one side of the midline in the occipital region.

Hydrocephalus—Treatment, ***continued.***

The dura mater is incised and any cortical vessels coagulated with the diathermy cautery. The thinned cortex is carefully incised and the ventriculoscope introduced into the posterior horn of the lateral ventricle. The fluid in the ventricle is aspirated and kept in a warm container so that it can be replaced at the end of the operation The choroid plexus is inspected, a fine cautery is passed and the majority of the plexus coagulated. Both plexuses can be removed at one sitting, but it is better to remove one at a time, an interval of one week intervening between the two operations. After the operation the head is fixed in a light plaster bandage to prevent the escape of cerebrospinal fluid.

External Hydrocephalus.—

This is a very rare condition. The fluid lies between the brain and dura. The brain is small and atrophic. Always associated with idiocy

TUMOURS CAUSED BY PROTRUSION OF THE BRAIN OR ITS MEMBRANES THROUGH THE SKULL

Congenital.—

MENINGOCELE —A protrusion of the meninges containing fluid.

ENCEPHALOCELE —A protrusion of brain substance through a congenital defect in the skull.

HYDRENCEPHALOCELE —A combination of the above, or a brain protrusion in which is a cavity communicating with the lateral ventricle

All these are large swellings which pulsate with all respiratory movements. Those containing brain also pulsate with heart movements. They usually spring from the root of the nose, the occiput, or the region of any of the sutures Round their broad base can be felt the bony margin of skull defect They are partly reducible, the reduction being accompanied by convulsions The skin over them is often thin and nævoid

TREATMENT is usually impossible In pure meningoceles excision may be possible Never attempt operative procedures where the condition accompanies a progressive hydrocephalus.

Acquired.—

1 SARCOMA OF THE BRAIN OR MENINGES.

2. HERNIA CEREBRI.—Occurs after · (*a*) Ineffectual operations for cerebral tumours; (*b*) Injuries or operations on the skull under septic conditions.

It is CAUSED by the increased intracranial pressure. The mass consists chiefly of œdematous granulations, but true brain substance may also protrude.

TREATMENT is chiefly: (*a*) Preventive, in making free outlet for septic discharges when operating for fracture of the skull; (*b*) Pressure must be used with great care; (*c*) Painting with absolute alcohol or collodion; (*d*) Covering with a protective shield; (*e*) Making free decompression elsewhere; (*f*) Removal is justifiable only when the hernia is due to a septic process which has come to an end.

INTRACRANIAL TUMOURS

Varieties.—Arise from: (1) Brain tissue; (2) Meninges; (3) Skull; (4) Miscellaneous.

1. FROM BRAIN TISSUE: GLIOMA—Belong to four groups:—
   - *a.* MEDULLOBLASTOMA.—Vermis of cerebellum in young children.
   - *b.* SPONGIOBLASTOMA MULTIFORME.—Adults 20–30 years; in the hemispheres.
   - *c.* OLIGODENDROGLIOMA —Frontal lobes of young men and women.
   - *d.* ASTROCYTOMA.—Vermis of cerebellam in children; hemispheres in adults 30–50 years.

   Pituitary tumours, acoustic tumours, ependymomata.

2. FROM MENINGES: MENINGIOMA.—Fifteen per cent of cerebral tumours Consist of whorls of endothelial cells. Have been termed endotheliomata, psammomata, and, when highly cellular, sarcomata. Many types are described—circumscribed, and diffuse or spreading. They do not infiltrate brain tissue, but may permeate the adjacent skull. They form no metastases, and are favourable for removal, but may be extremely vascular. Usually occur in the following sites: (*a*) Olfactory groove; (*b*) Suprasellar, (*c*) Sphenoid ridge; (*d*) Parasagittal meningioma.

3. FROM SKULL —Primary tumours of skull are rare (osteoma and sarcoma). The old sarcoma was usually a meningioma.

   Secondary tumours are carcinomata, melanomata, and sarcomata.

   Skull is commonly affected in multiple myelomatosis and von Recklinghausen's disease and Paget's disease, these are not strictly tumours.

4. MISCELLANEOUS.—Tuberculomata, gummata, secondary carcinomata, tumours of the vascular system, hydatid cysts, etc.

Symptoms.—The tumours listed above are extremely diverse in nature; each tends to have an individual life history and symptomatology. The tumours differ as widely as say the causes of intestinal obstruction, and so similarly symptoms differ, yet both groups progress to a common end—in the one obstruction, in the other increased intracranial tension. Some of the commoner symptoms are listed below:—

HEADACHE in a fixed locality, subject to great exacerbations and becoming steadily worse, it usually awakens the patient in the morning. But little amenable to therapeutic measures apart from operation.

VOMITING.—Constantly repeated without nausea or relation to food. Particularly severe in basic or subtentorial tumours. Both the headache and vomiting are often worse in the morning or after movement.

OPTIC NEURITIS.—Beginning as a congestion and then as a 'choked disc', and ending in atrophy. Most constant in basal growths, and is more severe in subtentorial tumours. The eye on the side of the tumour is affected first and to the most marked degree.

GIDDINESS, EPILEPSY, LOSS OF MEMORY, and other mental changes occur with different frequency, according to the regions affected.

MENTAL STUPOR, ending in COMA.

FOCAL SYMPTOMS are produced only when certain areas of the brain are affected (*see* p. 312). When a motor area is affected, localized convulsions followed by temporary local paralysis are the rule. The convulsions and paralysis tend to become more widely spread and more permanent.

Intracranial Tumours—Symptoms, ***continued.***

IN THE CEREBELLO-PONTINE TUMOURS there are paralysis of the third, sixth, and seventh nerves, with trigeminal anæsthesia or hyperæsthesia, and tinnitus succeeded by deafness, from involvement of the fifth and eighth.

Ventriculography; Encephalography; Arteriography.—These three procedures are purely diagnostic in nature. If the cerebrospinal fluid is in part replaced by air, X rays will then outline the ventricular system By this means the size, shape, symmetry, and patency of the ventricular and subarachnoid pathways can be demonstrated and valuable facts deduced. If intracranial tension is raised the air is introduced directly into the ventricle through a small burr hole in the skull; this procedure is ventriculography. If there is no tension the air may be introduced by lumbar puncture—encephalography. A radiograph of the skull while thorotrast is being injected into the carotid artery will outline the cerebral vascular tree (arteriography). In cases of suspected aneurysm or arterial malformation such a method is valuable. These three procedures involve inconvenience and in some cases much risk, and should only be undertaken by experts.

Treatment.—

MEDICINAL TREATMENT is useless, except in the case of gummata, where large doses of iodides should be given. Even in these cases if intracranial pressure is high decompression to save vision should be performed

OPERATIVE TREATMENT.—There is no one operation for cerebral tumour The procedure adopted will depend upon the life-history and situation of the tumour concerned. *Methods in use are*:—

1. OSTEOPLASTIC CRANIOTOMY over the hemispheres
2. PARTIAL CRANIECTOMY in the suboccipital region

with.

- *a. Removal of the Tumour*: Meningiomata, astrocytomata, many other gliomata, etc.
- *b. Biopsy of Tumour*: Medulloblastoma; certain glioblastomata multiformia
- *c. Partial Removal of Tumour*: Acoustic neurinomata; pituitary tumours; craniopharyngiomata, etc.
- *d. Internal Decompression*: Certain glioblastomata multiformia.

3. TEMPORARY or PALLIATIVE DECOMPRESSION

All these methods are combined where suitable with radiotherapy.

OTHER GENERAL PRINCIPLES.—The prevention of shock by:—

1. *Absolute Hæmostasis*.—This means slow, careful operating. Electrocoagulation, transfusions, etc.
2. *Careful Combinations of Anæsthesia*.—Local, basal, and occasionally inhalational anæsthesia.
3. *Relief of Pressure* by ventricular tapping.
4. Careful reconstruction of the wound tissues.

Pituitary Tumours.—Tumours of the pituitary gland differ from other intracranial tumours, in their relation to the general processes of body growth influenced by the pituitary internal secretion, and in their specially close effect upon vision owing to their proximity to the optic nerves.

FUNCTIONS OF PITUITARY.—Exercises controlling influence on carbohydrate metabolism and skeletal and sexual development, and regulates capillary tone.

HYPERPITUITARISM.—Produces gigantism if occurring in the young and acromegaly in the adult X rays may show expansion and irregularity in the sella turcica. Sexual hyperactivity. Polyuria, glycosuria. Melancholia.

HYPOPITUITARISM.—Produces stunted skeletal growth. Sexual functions undeveloped. Two common clinical types:—

1. *Dystrophia Adiposogenitalis* or *Fröhlich's Syndrome.*—Stunted, stupid, and fat children Genitals undeveloped
2. *Lorain Type* or *Ateleiosis.*—Stunted and sexually undeveloped, but mentally normal.

PATHOLOGY OF PITUITARY TUMOURS —

1 PITUITARY TUMOURS PROPER.—(*a*) Eosinophil adenomata, producing hyperpituitarism; (*b*) Chromophobe adenomata, producing hypopituitarism, (*c*) Basophil adenomata, producing Cushing's syndrome, i.e. high blood-pressure, cyanosis, hirsuties, and adiposity with impotence in the male and amenorrhœa in the female
2. EXTRA-PITUITARY TUMOURS —Cysts and meningiomas of Rathke's pouch.

SYMPTOMS.—Two classes: (1) Those due to mechanical causes (pressure effects); (2) Those due to disordered function.

1. MECHANICAL SYMPTOMS —
   - *a* Headache of bursting type
   - *b*. Bitemporal hemianopia.
   - *c*. Third-nerve palsy.
   - *d* Primary optic atrophy and blindness (cf ordinary cerebral tumours, which cause papillœdema and *secondary* optic atrophy).
   - *e*. Absorption of sella
   - *f*. Somnambulism and polyuria from pressure on floor of third ventricle.
   - *g* Paræsthesia of face (5th nerve).
2. SYMPTOMS DUE TO DISORDERED FUNCTION —
   - *a*. Hyperpituitarism from eosinophil adenomata.
   - *b*. Hypopituitarism from chromophobe adenomata and the extra-pituitary tumours.

OPERATIVE TREATMENT —

INDICATIONS.—Severe headache and progressive blindness.

METHODS.—

1. *Trans-sphenoidal Sellar Decompression* —Submucous resection of nasal septum exposing under surface of body of sphenoid. The sphenoidal cells are broken down and the gland exposed. This is rarely used except in some casos of intrasellar tumours
2. *Frontal Route* (Frazier).—An osteoplastic flap is turned outwards from the frontal region, and the diaphragma sellæ is reached by elevating the frontal lobe. This is always used now as method of approach. The tumour is curetted out, or, if cystic, is sucked out.

Intracranial Tumours, *continued.*

Congenital Cysts of the Suprasellar Region.—Arise in the pharyngeal diverticulum which gives rise to the pituitary. Best seen in children, usually multiloculated, frequently calcified, and may be of such a large size that they press on the floor of 3rd ventricle causing hydrocephalus.

CEREBRAL LOCALIZATION

The localization of function in different parts of the brain is shown by the disturbance which follows injury and disease. Such disturbance may be either an abnormal activity produced by an irritative lesion, or a diminished activity caused by a destructive lesion. Thus, in the case of motor centres, there may be convulsions or paralysis of muscles; and, with affections of sensory centres, abnormal sensations (e.g., tinnitus, flashes of light, tingling, or a sensory aura), or anæsthesia The nature of the localizing or focal symptoms depends on the situation of the local lesion more than upon its nature, so that quite different causes may produce the same focal symptoms. Further, there are certain general symptoms associated with intracranial disease which help to determine its nature and extent.

Local Lesions which may cause focal symptoms are: (1) Punctured wounds; (2) Depressed fractures; (3) Foreign bodies; (4) Meningeal hæmorrhage or inflammation; (5) Scar tissue associated with the cerebral cortex; (6) Abscess; (7) Tumour.

General Symptoms associated with intracranial disease and usually accompanying cerebral compression are as follows:—

HEADACHE of cerebral origin Severe, worse on movement, and usually fixed to a special locality of the head It is probably caused by pressure on the dura mater, and is roughly proportionate to (*a*) the intracranial tension, and (*b*) the direct implication of the meninges by disease It must be distinguished from the following groups:—

Toxic.—Associated with any condition of fever or toxæmia

Renal.—This is probably a cerebral condition caused by œdema of the brain. The conditions of the heart and urine give clues to its nature.

Reflex.—From disease of the sense organs. Disease of the nasal sinuses, glaucoma and iritis, otitis, and dental caries are examples of these.

VOMITING.—Usually sudden, projectile, and unaccompanied by nausea. It is worse after movement

VERTIGO.—Especially associated with lesions of the eighth nerve, the mid-brain, and the cerebellum

OPTIC NEURITIS —Occurs in two stages.

Choked Disc is the congestion of the retina and œdema of the optic papilla resulting from increased intracranial pressure It is usually bilateral, but the eye on the side of the lesion may be affected earlier or to a higher degree than the opposite, though this cannot be relied upon.

Neuritis and Atrophy.—True neuritis may be caused by long-continued pressure or congestion, or atrophy may be the primary change. In the latter case a lesion at the base of the brain is indicated.

Blindness may not be present, even with a high degree of choked disc or neuritis.

VISUAL FIELDS.—Interference with the visual pathway causes changes in the visual fields, revealed by perimetry. Accurate diagnosis may be made in this manner.

Localization of Function in the Cerebral Cortex.—The following paragraphs are in the main based on animal experimentation and isolated human observation. Thus the observed effects are usually the results of dramatic experiments like sudden ablation or stimulation. These all-or-none phenomena cannot be compared with the gradual interference produced by a tumour or chronic infection. Hence the occasional remarkable dissimilarity in symptomatology which may be found.

Each cerebral hemisphere is functionally related to the opposite side of the head and body. Centres for voluntary movements are more sharply marked than those for sensation

THE MOTOR CENTRES (*Fig.* 131) lie in the pre-central or ascending frontal gyrus immediately in front of the fissure of Rolando. They occupy the following position from above downwards:—

Foot Knee Hip Trunk	upper third	Shoulder Elbow Wrist Fingers and thumb	middle third	Neck Nose Lips Jaw	lower third

The centres for the trunk and for the neck are opposite the genua or bends taken by the Rolandic fissure, and serve to separate the centres for the leg, arm, and face The centres for the tongue, palate, larynx, and vocal cords lie close together above the front of the main limb of the Sylvian fissure

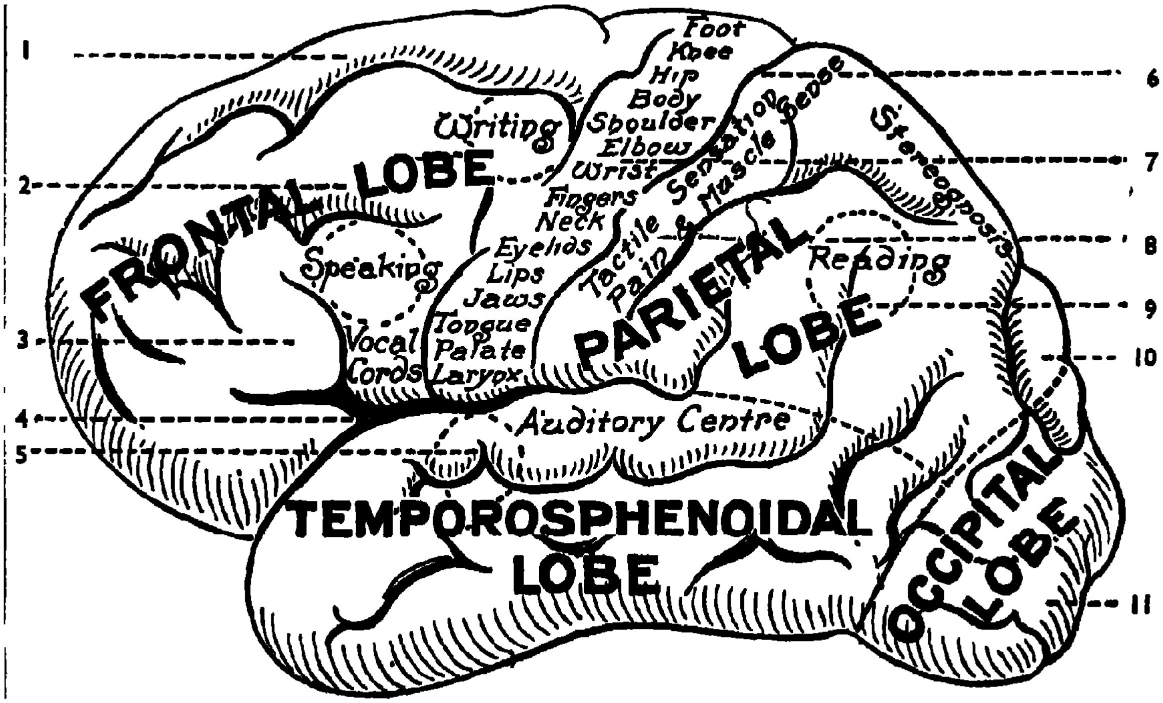


Fig. 131.—Localization of function in cerebral cortex. 1, Upper frontal gyrus; 2, Middle frontal; 3, Inferior frontal; 4, Fissure of Sylvius, 5, Word hearing; 6, Fissure of Rolando; 7, Precentral gyrus; 8, Post-central gyrus; 9, Angular gyrus; 10, Parieto-occipital fissure; 11, Occipital lobe.

NOTE —The leg centres are rather larger than those for the arm, but in this lateral view are necessarily foreshortened.

Localization of Function in the Cerebral Cortex, ***continued.***

THE MOTOR SPEECH CENTRE is in Broca's convolution on the left side (this applies to right-handed persons; in left-handed persons it is in the right hemisphere). This lies in the angle between the vertical and posterior horizontal limbs of the Sylvian fissure, and is the posterior part of the third frontal convolution.

THE MOTOR WRITING CENTRE is at the posterior part of the second left frontal convolution, just in front of the motor centre for the hand.

SENSORY CENTRES.—

COMMON TACTILE SENSATION is localized in the postcentral or ascending parietal gyrus just behind the Rolandic fissure. The sensory centre for each part of the body is just behind the corresponding motor centre.

CENTRES FOR PAIN AND MUSCULAR SENSATION are not represented in the cortex. Probably in the optic thalamus.

STEREOGNOSTIC SENSATION is localized in the upper parts of the parietal lobe. Stereognosis is the power of recognizing the shape and size of objects by touch.

VISUAL CENTRE is in the occipital lobe, with which the angular gyrus is associated The left half of each retina is associated with the left occipital lobe, and vice versa

READING CENTRE is in the left angular (parietal) gyrus

AUDITORY CENTRE is in the superior temporal gyrus

THE SPEECH-HEARING CENTRE corresponds to the left auditory centre.

OLFACTORY AND TASTE CENTRES probably lie near tip of the temporal lobe at its mesial aspect, i.e , the uncinate gyrus.

The Symptoms arising from Local Brain Lesions.—

MOTOR.—Convulsions or paralysis

CORTICAL LESIONS usually cause monoplegia associated with convulsions.

CAPSULAR LESIONS cause hemiplegia without convulsions.

PONTINE LESIONS cause 'crossed paralysis,' i e., same side of the face, opposite side of the body

BIRTH PALSIES AND SPINAL PALSIES are usually spastic diplegia or paraplegia.

SPASTICITY associated with paralysis indicates a lesion of conducting tract and not motor centre

CONVULSION associated with paralysis indicates a lesion of the motor centre.

SENSORY.—

PARALYTIC.—Anæsthesia of various kinds. It is usually very fleeting. It is most marked at the extremities farthest removed from the brain, and gradually shades off into areas of normal sensation.

IRRITATIVE.—Some abnormal, often painful, sensation, either special or general. Such are the auræ which precede epileptic fits.

The Regions of the Brain associated with Focal Symptoms.—

ROLANDIC AREA.—

IRRITATIVE LESIONS cause Jacksonian convulsions, which begin in certain muscles and spread in the order of the arrangement of the motor centres to other muscles. Consciousness may not be lost until half the body is affected.

Sensory Symptoms, as stated above, are very transient with cortical injuries. More permanent anæsthesia is produced by subcortical lesions (e.g., of the internal capsule), and is always associated with some paralysis.

FRONTAL LOBE.—(Clinically this term excludes the ascending frontal gyrus, which constitutes the motor area.)

The Front and Upper Parts of the Frontal Lobe (prefrontal area) are unexcitable, and extensive lesions may be quite latent. Or they may cause insidious changes of character, loss of memory, apathy, loss of power of inhibition. Most marked in left prefrontal lesions.

The Postfrontal Region below contains the motor speech centre on the left side Lesions of this cause aphasia.

PARIETAL LOBE.—(Apart from the postcentral Rolandic gyrus.)

Lesions of the Angular Gyrus on the left side cause word blindness.

Of the Upper Parietal Lobe.—Loss of stereognostic sense, i.e., the power of recognizing objects by tactile sense.

OCCIPITAL LOBE —Destructive lesions cause homonymous hemianopsia (i.e., blindness of that half of the retina of both eyes which is on the same side as the lesion) This is permanent if the angular gyrus is destroyed simultaneously. The pupil reflex is not lost. **Word Blindness** results from lesions of the left angular gyrus

TEMPORAL LOBE —**Word Deafness** results from lesions of the superior left temporal gyrus

Olfactory and Gustatory symptoms are caused by lesions of the uncinate gyrus

CAUDATE AND LENTICULAR NUCLEI AND INTERNAL CAPSULE.—Hemiplegia, with some sensory loss.

OPTIC THALAMUS —Contralateral athetosis and chorea, paræsthesia, and hemianopsia

CRUS CEREBRI.—Hemiplegia, with oculomotor paralysis and loss of pupil reflex of the eye on the same side as the lesion.

CORPORA QUADRIGEMINA —Ophthalmoplegia, reeling gait, vertigo, tendency to fall backwards.

PONS —Paralysis of the face on the side of the lesion and of the limbs on the opposite side.

CEREBELLUM.—Instability of station and locomotion, with coarse ataxia accompanying voluntary movements.

Middle Lobe.—The symptoms are bilateral. There is a tendency to fall backwards or forwards

Lateral Lobe.—Weakness of the movements of the arm on the same side. The head is bent to the side of the lesion and the face turned away from it. In walking there is a tendency to rotatory deviation, turning or falling towards the affected side. There may be conjugate deviation of the eyes and nystagmus towards the side of the lesion. (The eye movements are variable in cerebellar disease. A lateral excitation causes deviation of the eyes to the same side. A lateral paralytic lesion may cause deviation of the eyes to the opposite side. But often the lesion causes no eye symptoms.)

INTRACRANIAL INFLAMMATION

VARIETIES.—EXTRADURAL (abscess or pachymeningitis); MENINGITIS (acute or chronic, diffuse or local); INFECTIVE THROMBOSIS of the sinuses; ABSCESS in the brain substance; DIFFUSE ENCEPHALITIS.

Extradural Abscess.—Pus collects between bone and dura mater.

CAUSES.—Septic open fracture. Septic scalp wound and osteomyelitis. Contusion followed by auto-infection. Middle-ear disease

SIGNS AND SYMPTOMS.—Symptoms of suppuration. Compression of the brain. Unhealthy wound with dead bone or œdematous patch on scalp (Pott's puffy tumour) Localized headache and tenderness. Paralysis or spasm if over motor area.

TREATMENT —Trephine, evacuate, and drain Chemotherapy.

MENINGITIS

Pachymeningitis.—Local thickening of dura Probably follows contusion or fracture Fixed headache, with possibly Jacksonian epilepsy.

Acute Meningitis may be primary and idiopathic, or secondary and septic.

Primary Idiopathic Meningitis occurs as epidemic cerebrospinal fever, due to *Diplococcus intracellularis* In infants sporadic cases of posterior basic meningitis occur, due to same organism

SYMPTOMS —Acute onset, with headache and rigors, vomiting, photophobia, and optic neuritis Marked retraction of the head and spasms of the limbs If not immediately fatal it sometimes leads to acute hydrocephalus or epilepsy

TREATMENT by repeated lumbar puncture. Drugs of the sulphonamide group are extremely effective

Secondary hydrocephalus may be relieved by major neurosurgical procedures

Acute Septic Meningitis.—Infection by pyogenic cocci, those secondary to fractures of the vault being usually streptococcal, and those following basal fractures pneumococcal, from infection through nasal sinuses

CAUSES —Septic wounds of the scalp or orbit. Suppuration in nasal cavities or sinuses Infection from pharynx or fauces Extension from otitis media

SYMPTOMS —Rapid onset of symptoms forty-eight hours after injury. Severe headache. Vomiting of cerebral type. Photophobia Cerebral irritability, followed by delirium with sharp cry, and later by signs of cerebral compression Spasm and twitchings of muscles. Retraction of the head. High temperature, initiated by a rigor

IN MENINGITIS OF THE CONVEXITY.—Convulsions affecting special groups of muscles.

IN MENINGITIS OF THE BASE.—Optic neuritis—Strabismus, from paralysis of motor nerves—Marked retraction of the head

LUMBAR PUNCTURE is of great value both for diagnosis and treatment Performed between the 3rd and 4th lumbar spines. The cerebrospinal fluid escapes with a jet as if under pressure. It contains many lymph-cells—chiefly polymorphs in septic meningitis and lymphocytes in tuberculous cases. Also the causative bacteria, from which a vaccine may be prepared. In posterior basic cases the fluid is often sterile.

Acute Septic Meningitis, *continued.*

ANATOMY.—Vascular engorgement of dura and pia mater, cerebral substance, and choroid plexuses. Arachnoid is thick and opaque. Turbid serum or pus occupies the subarachnoid space. Convolutions are flattened. Ventricles are distended.

TREATMENT.—Rest and treatment of the general febrile condition. Cold to the head. Repeated lumbar puncture. Cistern puncture. The sulphonamide group of drugs often prove very useful.

Chronic Traumatic Meningitis.—

CAUSED BY a local head injury.

CONSISTS OF a thickened patch of membranes May be adherent to the bone above or to the brain below.

SYMPTOMS —Headache, constant, fixed, and localized—Local tenderness —Possibly traumatic epilepsy—Mental instability—Alcoholism.

TREATMENT —Prolonged rest Absence of excitement. Abstinence from alcohol When preceding history of injury is definite, and no relief from simpler measures, exploratory trephine for subdural hæmatoma is advised.

Chronic Syphilitic Meningitis.—A severe headache is associated with a variety of mental symptoms and cranial nerve paralyses. The symptoms are intermittent in severity

TREATMENT —Six weeks' medicinal treatment should be tried first If this does not effect a great improvement, an attempt should be made to remove any localizable thickenings, or to perform a decompressive operation if intracranial tension is high

INFECTIVE THROMBOSIS OF THE CEREBRAL SINUSES

CAUSES —Middle-ear disease (lateral sinus) Septic wounds of scalp or orbit. Septic states of nose or pharynx Erysipelas or cellulitis of face or scalp Osteomyelitis of cranial bones

VARIETIES.—Lateral sinus—much the commonest—infected from the ear Cavernous sinus—next commonest—infected from nose, face, or orbit. Longitudinal sinus—rare—infected from the scalp, face, or nose.

ANATOMY —Sinus is plugged by soft, adherent, septic blood-clot. Portions of the infected clot form septic emboli Emboli cause pulmonary infarctions and abscesses. Inflammation of the bone, meningitis, or abscess are probable complications

SYMPTOMS AND SIGNS —Existence of some primary septic focus.

HEADACHE.—Sudden and severe. Localized tenderness over the affected sinus

REPEATED VOMITING

TEMPERATURE rises to 103°–105°, with a bad rigor; continuous, with remission and REPEATED RIGORS. Dyspnœa and cyanosis are marked during the rigors.

PULSE is full, slow, and compressible, but becomes rapid during the rises of temperature.

LOCAL PAIN and other signs in the lungs may point to the existence of INFARCTIONS.

MENTAL CONDITION —Drowsy or delirious.

OPTIC NEURITIS AND STIFFNESS OF NECK MUSCLES point to meningitis.

Infective Thrombosis of the Cerebral Sinuses, ***continued.***

IN LATERAL SINUS THROMBOSIS.—Discharge from the ear ceases. Tender swelling over the jugular vein Dusky congestion of the side of the face. An abscess may form in the neck Rarely the nerves emerging from the jugular foramen may be affected, producing hoarseness, dysphagia, dyspnœa, or slow pulse (jugular bulb syndrome).

IN CAVERNOUS SINUS THROMBOSIS.—Marked exophthalmos, with great orbital congestion. Chemosis, choked disc, retinal hæmorrhages, ocular paralysis, with hyperæsthesia of first division of fifth nerve and occasional herpes May become double by extension of the thrombosis across the pituitary sinuses.

IN SUPERIOR LONGITUDINAL SINUS—Turgescence of the scalp and forehead Tenderness over the sinus. Convulsions of one or both legs.

TREATMENT.—

For Lateral Sinus.—Trephine on Reid's base-line, ¾ in. behind external auditory meatus. Tie internal jugular vein first if diagnosis is certain. Turn out the clot. Perform a radical mastoid operation if necessary. Plug

For Longitudinal Sinus—Establish drainage from focus of infection. Treat the cause; also chemotherapy should be instituted. Repeated small whole blood transfusions are of value.

ABSCESS OF THE BRAIN

CAUSES.—

1. **Traumatism**, e g , fracture or penetrating wound.
   - Early after injury—superficial, contiguous to bone, really an encephalitis
   - Late after injury—deep in white matter of the frontal or parietal lobes.
2. **Infection from Neighbouring Septic Foci**—
   - Middle-ear disease, usually of the chronic type · Generally in cerebellum or temporosphenoidal lobe, the latter being about twice as common as the former Sepsis may spread by direct continuity through bones and membranes More commonly spreads by vessels and occurs beneath the surface
   - Disease of frontal, sphenoidal, or ethmoidal sinuses.
   - Thrombosis of venous sinuses
3. **Blood Infection in Pyæmia** or exanthemata.
   - Septic lung diseases, e.g , gangrene or bronchiectasis.
   - Osteomyelitis.
   - Septic endocarditis.
4. **Tubercle** may cause a chronic abscess.

ANATOMY.—Generally single and often of a chronic type having a well-marked capsule May burst into ventricles or subarachnoid space, or cause œdema of brain The pus is usually offensive, and contains the usual pus cocci or pneumococci.

SYMPTOMS.—

Discharge from the ear ceases, if of otitic origin.

Headache.—General at first; fixed and localized later—often intermittent.

Mental Condition.—Drowsiness, with irritability.

Temperature normal, or often subnormal.

VOMITING is frequent. Anorexia, malaise, and constipation.

OPTIC NEURITIS.

COMPRESSION SIGNS.—Slow or intermittent pulse. Laboured breathing —Cheyne-Stokes type later. Torpor is succeeded by coma. Pupil on the side of lesion is dilated and fixed.

LATENCY, with absence of some or all symptoms, is common.

FOCAL SIGNS (rare).—

Temporosphenoidal.—Paralysis or spasm of the opposite side of face; aphasia if the left side is affected.

Cerebellum.—Giddiness and tendency to fall towards the lesion. Nystagmus and deviation of eyes to the same side (*see* p. 313—FOCAL SYMPTOMS IN DISEASE OF CEREBELLUM). Paralysis of the arm on the same side.

TREATMENT —Falls into two groups: (1) Localization of the abscess (history; neurology; ventriculography); (2) Relief of intracranial tension (lumbar puncture, decompression—tapping). Most abscesses start as acute or subacute foci of semi-diffuse inflammation, and their symptoms are due to œdema. If the patient can be tided over the acute stage the abscess may become thick-walled and chronic, and can be extirpated as a tumour as advised by Cushing and Horrax. Alternatively abscess may be marsupialized.

Abscesses which are a definite extension of mastoid infection are usually treated by evacuation along the route of infection, i.e., via a radical mastoid operation.

OTITIS MEDIA

Varieties.—Catarrhal, acute purulent, and chronic purulent.

Causes.—Pharyngitis—Pharyngeal catarrh (spreads up the Eustachian tubes)—Tonsillitis—Scarlet fever and other fevers—Diphtheria—Adenoids.

Symptoms.—

Great pain on onset, relieved when the drum perforates. Temperature raised to 100°–105° F.

Deafness—Usually partial only—Tuning-fork can be well heard if applied to the bones—Tuning-fork can be better heard in the affected ear when held to the vertex.

Tinnitus.

Purulent discharge from the meatus: only after the drum has perforated.

Signs.—

BEFORE PERFORATION OF THE DRUM —Drum is first red, and then has lost its lustre, and is abnormally bulged outwards. It does not move on swallowing.

AFTER PERFORATION.—On clearing away pus from meatus, tympanum is seen to be perforated In old cases the tympanum is destroyed, and bare bone or the ossicles may be seen or felt. On politzerization air escapes from the meatus if the Eustachian tube is patent.

Treatment of uncomplicated cases.—

CATARRHAL OTITIS.—Usually associated with pharyngeal catarrh or adenoids. Treat the pharyngeal catarrh with astringent gargles or sprays, e.g., protargol gr. iv ad ℥j. Remove adenoids when attack has subsided. Syringe meatus with warm boracic lotion. For acute pain of 'earache', leech in front of the tragus, and hot sand-bags.

Otitis Media—Treatment, *continued*.

ACUTE PURULENT OTITIS.—

Before Perforation, or if the perforation is minute, heat and chemotherapy. Free incision of the tympanic membrane from the centre downwards, followed by boracic irrigation. General treatment of the febrile condition.

After Perforation, when free discharge has taken place, mop out the meatus and then dry and instil boracic powder.

COMPLICATIONS OF PURULENT OTITIS MEDIA

Most commonly seen with the chronic cases. (*Fig.* 132)

EXTRACRANIAL.—Eczema of the meatus. Boils and deep inflammation of the meatus. Suppurative arthritis of temporo-maxillary joint.

CRANIAL.—Ankylosis or necrosis of the ossicles. Necrosis of parts of the temporal bone. Polypi (granulations) in place of the destroyed drum. Facial paralysis from pressure on the seventh nerve. Mastoiditis. Labyrinthritis.

INTRACRANIAL COMPLICATIONS.—Extradural abscess—Meningitis—Thrombosis of the lateral sinus—Cerebral abscess in temporosphenoidal lobe or cerebellum

Acute Mastoiditis.—

SYMPTOMS.—Intense pain of a constant character. Tenderness behind the ear over the mastoid process Redness and œdema over the mastoid. Auricle is displaced forwards, outwards, and downwards Eventually a fluctuating abscess forms behind the ear General febrile symptoms: temperature 102°–104° F Sagging of the posterior wall of the meatus is a reliable sign

Rigor may occur (but repeated rigors point to sinus thrombosis).

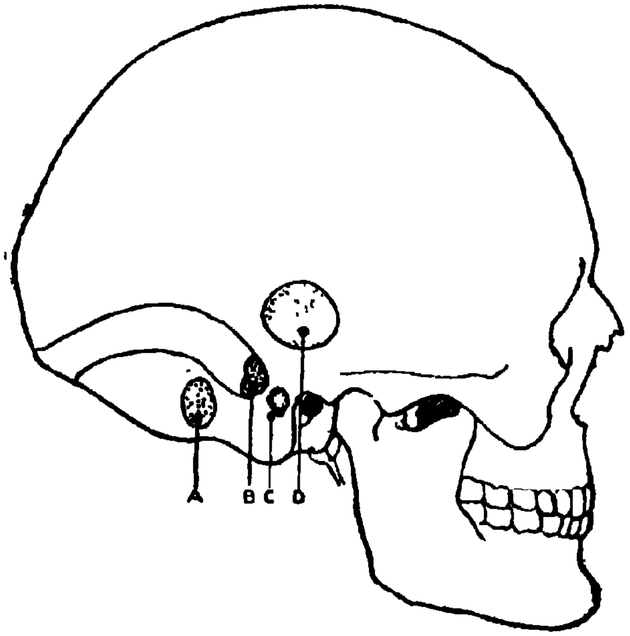


Fig. 132.—Complications of middle-ear disease. A, Cerebellar abscess; B, Lateral sinus thrombosis, C, Suppuration in mastoid antrum; D, Temporo-sphenoidal abscess.

TREATMENT.—

In Early Stage before the diagnosis is certain.—Hot fomentations, or hot pad over the ear.

When the Diagnosis is Clear.—A cortical mastoid operation is required. Through a curved incision over the mastoid the antrum is opened and then systematically all mastoid air-cells are removed. This may involve extension forward into the zygoma, upward to the skull base, backward to the sinus and beyond, and the whole of the process to its tip. Drainage. This is the cortical or Schwartze modified operation.

Chronic Mastoiditis.—This seldom spontaneously resolves. It is a potential menace because of intracranial complications. Therefore some variety of operation is indicated. The modified or conservative radical mastoid operation if done early will often suffice and conserve hearing to some extent. The ossicles are spared, the mastoid is drained via the external auditory meatus. In worse cases a complete radical operation involves removal of the ossicles.

Intracranial Complications of Otitis Media.—

DIAGNOSIS.—In all the following occur. Marked cerebral symptoms. Acute headache. Coma or delirium Purulent discharge from the ear becomes scanty or ceases.

SINUS THROMBOSIS.—Repeated severe rigors. Temperature rises to 103°–105° F., but falls between the rigors Patient is blue and collapsed. Tenderness or swelling over the internal jugular vein

MENINGITIS —Initial rigor, with continuous high temperature. Retraction of the head. Optic neuritis. Intolerance of light and sound. Rapidly developing coma.

SUBCRANIAL ABSCESS (pus between the dura and the petrous bone).—Severe headache. Gradual cerebral compression. Irregular fever. Absence of the signs characteristic of thrombosis, meningitis, or abscess.

CEREBRAL ABSCESS.—Temperature is normal or subnormal. Constant headache, merging into coma. Slow pulse. Slow respiration—Cheyne-Stokes later Optic neuritis and paralysis of pupil on affected side.

CEREBELLAR ABSCESS.—In addition to the above, may be nystagmus. Giddiness, with tendency to fall to affected side.

TREATMENT OF INTRACRANIAL COMPLICATIONS OF OTITIS.—First expose and ligature the internal jugular vein in the neck (if sinus thrombosis is clear). Perform radical mastoid operation and follow track to abscess, removing bone upwards and forwards for temporo-sphenoidal abscess, or backwards for sinus thrombosis and cerebellar abscess. (*See also* section on Abscess of the Brain, p. 316)

CHAPTER XXVII

DISEASES OF THE LIPS AND JAWS

THE LIPS

Macrocheilia or Thick Lips.—

1. CONGENITAL.—Lymphangiectasis—Clear lymph-vesicles can be seen on the surface—Generally affects the lower lip.
   TREATMENT.—By excision of a wedge-shaped piece.
2. TUBERCULOUS.—In children and young adults—Generally the upper lip—May be associated with tuberculous disease of the nose—Fibrous and œdematous thickening round fissures
3. SYPHILITIC.—Fibrous hypertrophy in tertiary disease.

Ulcers of the Lips.—

1. SIMPLE.—Cracks and fissures result from cold in those with bad circulation. Chilblains and ulcerated chilblains arise in the same way.
2. HERPES.—Generally unilateral. Associated with catarrh or pneumonia. Vesicles become pustular and then break.
3. TUBERCULOUS —Either chronic indurated fissures causing macrocheilia; or definite lupus, with clear tubercles and destructive ulceration.
4. SYPHILITIC.—
   PRIMARY CHANCRE —Usually the upper lip. Caused by kissing person with secondary lesions of mouth, or using pipe or cup contaminated by such a patient Flat ulcer on an infiltrated base. Skin more involved than the mucous membrane. Glands enlarged and massed together under the jaw (not so discrete as in genital chancre).
   SECONDARY.—Mucous tubercles and shallow, painful ulcers.
   TERTIARY —Deep serpiginous ulceration destroying the lip—glands not enlarged. Or general fibrous hypertrophy (macrocheilia).
   INHERITED.—Cracks and fissured ulcers radiating from the angles of the mouth. Leave permanent scars and contraction.
5. MALIGNANT.—
   EPITHELIOMA.—95 per cent are in men—often pipe smokers. Much commoner in lower than upper lip. Begins as crack, ulcer, or wart. Causes an ulcer or warty growth—indurated and everted margin, and indurated base. Submental and submaxillary glands become hard and enlarged comparatively late, i.e., after three to nine months.

   Much slower in its development than epithelioma of the tongue or fauces.

   Diagnosis should always be made certain in doubtful cases by removal and microscopical examination.

   Treatment.—

   a. Excision with a ½ in. margin, followed by a block dissection of both submaxillary triangles.

   b. Radium. Implantation of radium tubes in and round the growth. Radiation of the submaxillary regions by a moulded collar containing properly spaced radium needles.

c. Low-voltage contact X-ray therapy to the affected lesion, with block dissection of the glands of neck.

Death occurs from septic ulceration of the glands in the neck.

Tumours of the Lips.—Besides the ulcers given above, the following occur:—

WARTS.—Remove for fear of malignant growth, and examine with microscope.

NÆVI.—Dissect out or destroy by electrolysis.

CYSTS.—Caused by retention in the labial mucous glands. Small round fluid swellings containing glairy mucous fluid. Dissect out.

Hare-lip.—Congenital cleft in upper lip. Usually single and unilateral, left-sided, and in boys. Often double and bilateral. In simple cases the soft parts only are affected. In others the alveolus of the jaw or the palate may be cleft. When complete it extends into one nostril. Commoner on left side.

In all cases the nose is broad and flat. In double cases the bone between the clefts projects forward like a proboscis. Often associated with other deformities, e.g., talipes, etc. If the alveolus is involved, the cleft generally goes between the central and lateral incisor.

CAUSED by a failure of union of the mesial nasal process with the maxillary process superficially, and the lateral nasal process more deeply.

EFFECTS.—Interference with suckling, and with speech. Deformity, which gets worse with age.

TREATMENT.—Operate at six weeks to three months.

FOR SINGLE HARE-LIP (*Fig.* 133).—Separate central parts of the lip from bone beneath. Pare the edges. Leave single or double flap of red margin, so that when sewn together there will be an allowance for contraction. Two deep stitches to take off tension. Many fine horsehair stitches to edges. Plaster strapping across the face to relieve tension Put arms into splints. Feed by spoon, or better, nasal tube. Allow to suck at end of one week.

FOR DOUBLE HARE-LIP —

Treatment of os incisivum when projecting forwards: Refresh the anterior end of the nasal septum, push back the proboscis and sew to the septum.

Treatment of cleft: Separate lip from the underlying bones Pare both edges of both clefts. Sew outer margins of both clefts together below central part, which takes no part in margin. Leave a flap from red margin of each outer margin of each cleft.

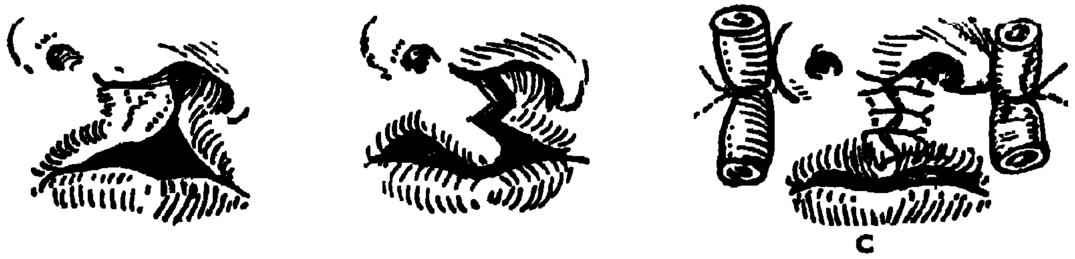


Fig. 133.—Repair of single hare-lip. A, Complete single hare-lip. B, Showing lines of incision. C, Sutures in place. One deep tension stitch is tied over rubber tubing.

CLEFT PALATE

A congenital defect in the union of the two halves of the palate.

Degrees.—(1) Bifid uvula; (2) Cleft soft palate; (3) Soft and hard palate cleft as far forward as the anterior palatine foramina; (4) Including the alveolar border of the maxilla and lip, i.e., combined with a double hare-lip.

Varieties.—

1. Mesial—the cleft is in the mid line, and the nasal septum is above and ununited to any part of the cleft.
2. Bilateral—the nasal septum grows down and divides the cleft into two (*Fig.* 134).
3. Unilateral—the nasal septum unites with one side of the cleft. The cleft is usually on the left side (*Fig.* 135).

Development of Alveolar Cleft Palate.—In man the premaxillæ are commonly rudimentary pieces of bone which are attached to the apex of the vomer. They take no part in the formation of the face, and bear no teeth. The maxillæ, on the other hand, send two processes inwards, which surround the premaxillæ, excluding the latter from the face; these are:—

1. The incisor process in front, which meets its fellow and bears the incisor teeth.
2. The palatine process behind, which forms the hard palate.

Between these two the anterior palatine foramen is left, in the inner wall of which the rudimentary premaxillæ remain.

In simple cleft palate, the palatine processes of the maxillæ are defective.

In alveolar cleft palate, the incisor processes of the maxillæ are defective, and the premaxillæ grow forward into the face, as is the rule in lower animals. The alveolar cleft is then the space between the abnormal premaxilla and the undeveloped incisor process of the maxilla.

In the most extreme cases, the premaxillæ bear all four incisor teeth, and there is a cleft on each side of them separating them from the maxillæ bearing the canines.

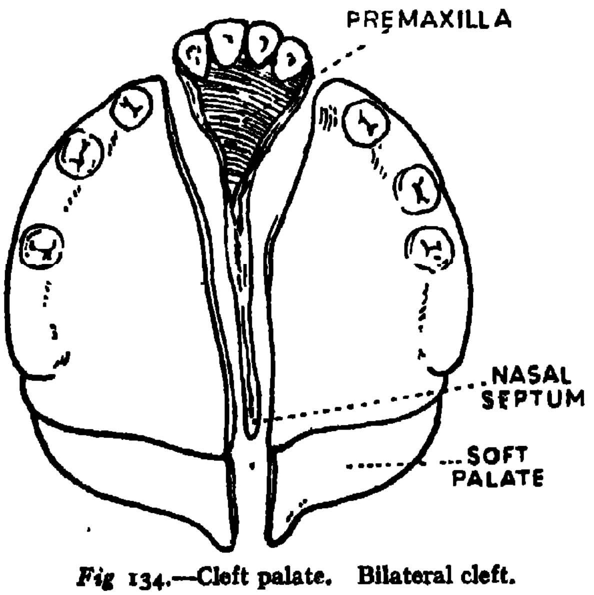


Fig 134.—Cleft palate. Bilateral cleft.

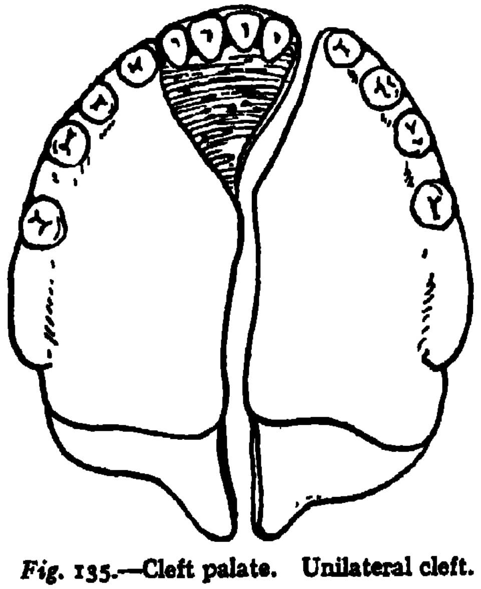


Fig. 135.—Cleft palate. Unilateral cleft.

In less-developed cases the premaxillæ bear only one incisor, and the incisive process of the maxilla bears the other, and the cleft comes between the central and lateral incisors.

Anatomy.—Apart from gross defects and their varieties above-mentioned.

The steepness of the palatal shelves varies from being nearly horizontal to being inclined at about 45°.

The soft palate is always undersized and is not long enough to fold up against the posterior pharyngeal wall. Sewing the two sides of the palate together further shortens it. Hence the essence of modern methods of repair is to free the soft palate from the hard and to shift it backwards

Effects.—Sucking is impossible, and the infant must be spoon-fed.

Defective speech is caused by the impossibility of closing the oral from the nasal cavity. Thus explosive sounds, e g., the letters *T*, *D*, *P*, *B*, *F*, *V*, become *M* or *N*, and gutturals, e g., *K* and *G*, become *H*

Physiology of the Palate.—The palate forms a partition separating the mouth from the nose The soft palate should be able completely to close the oral from the nasal pharynx This is done not merely as a flap valve, but by the active contraction of the muscles surrounding the palato-pharyngeal region These are mainly the palato-pharyngei behind and the tensores and levatores palati in front The contraction of the palato-pharyngei lifts up a transverse fold across the back of the pharynx (Passavant's ridge), whilst the other muscles pull the soft palate against this structure. It is in fact a sphincteric action.

Operative Treatment.—No dental plate can provide the mobile mechanism necessary to close the mouth from the nose. *The old operations* failed to cure the condition because, although they provided a partition bridging the gap, they did not provide a long enough soft palate to close against the pharynx. Thus the classical operation of Langenbeck, in which the edges of the cleft were sewn together, and the Lane operation, in which flaps of mucous membrane were turned over, both left the patient quite unable to close the oral from the nasal pharynx, and thus no better off in the matter of speech. They are now only of historical interest. Brophy's operation crushed together the maxillæ and fixed them by wires. It is destructive and dangerous *All modern methods have as their aim* (1) Mobilization and union of the soft palate, (2) Some device for bringing the soft palate back so that it may be able to meet the posterior pharyngeal wall. There are three chief types of these methods:—

GILLIES' —The soft palate is divided from its attachment to the hard on each side and then the two halves are sewn together This leaves a larger gap in the hard palate, which is closed by a dental obturator.

WARDILL'S (Modification of Veau's).—

a. Begins by a pharyngoplasty—a transverse incision across the posterior pharyngeal wall through the superior constrictor muscle, sewn together longitudinally

b. Long lateral incision at the junction of alveolus and palate Hamulus is broken off. Division of posterior palatine artery. Separation of mucoperiosteum from the hard palate.

c. Oblique incision on each side so as to form two anterior and two posterior flaps.

Cleft Palate—Operative Treatment—Wardill's, *continued.*

d. Suture. Median edges of cleft sewn by double layer (nasal and oral). Anterior flaps sewn together and to back of posterior flaps. (*Figs.* 136, 137, 138.)

DENIS BROWNE'S.—Two stages:—

a Removal of tonsils. Division of posterior palatine artery.

b. Separation of soft palate from the hard, and suture. Long lateral incisions from anterior pillar of fauces to incisor tooth. Breaking off hamulus (to free the tensor palati). Separation of mucoperiosteum from hard palate. Division of attachment of soft palate to the posterior margin of the hard. Suture in double rows. (*Figs.* 139, 140.)

GENERAL REMARKS.—

The modern operation is done between the age of one and two. The anæsthetic is intratracheal.

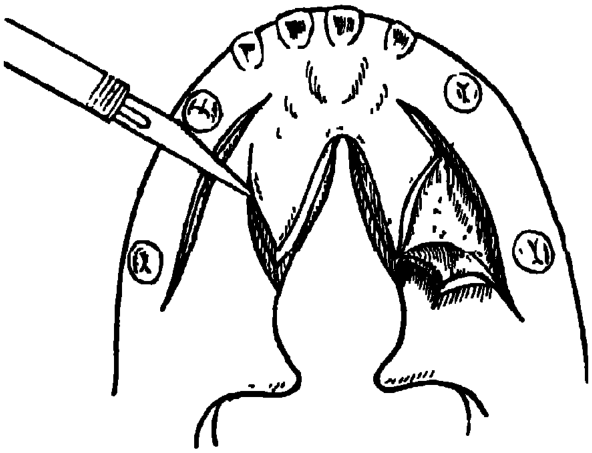
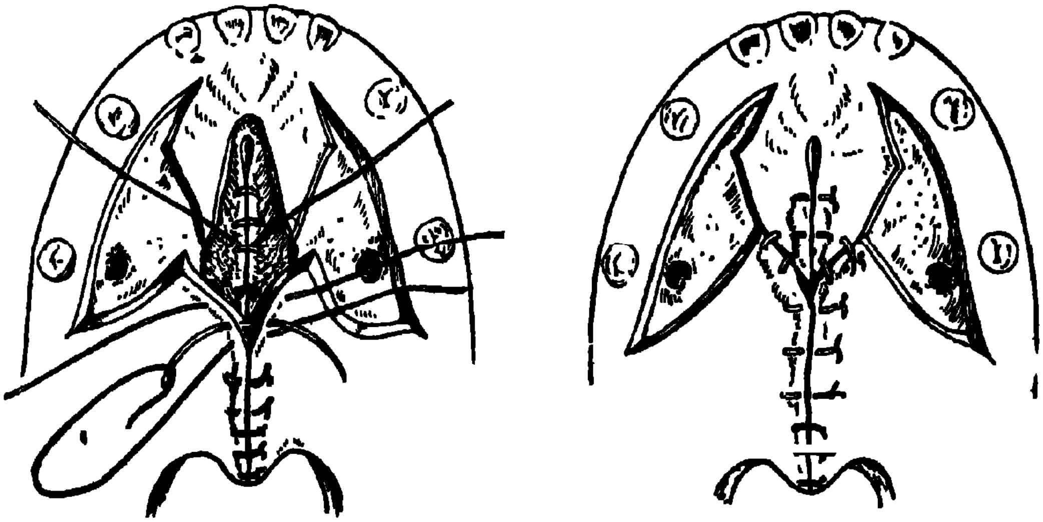


Fig. 136.—Cleft palate. Wardill's operation



Figs. 137, 138.—Cleft palate. Wardill's operation.

As a result the restoration of normal speech is the rule and not the exception as it used to be in the old methods.

After-treatment.—Feed by nasal tube for one or two weeks. It is wise to have educated the child to this before the operation.

THE JAWS

Alveolar Abscess.—Results from a carious tooth. May open on the surface of the alveolus, externally, on the palate, or into the antrum. Often causes periostitis, rarely necrosis.

TREATMENT.—By removal of the tooth and opening the abscess.

Epulis.—A growth from the alveolar periosteum or periodontal membrane.

VARIETIES.—False and true.

False.—Granulomatous tissue produced by irritation of dead or carious tooth.

True.—Is a new growth May be:—

1. *Fibrous*.—Forms polypoid growth.
2. *Fibrosarcomatous*
3. *Myeloid*.—A benign giant-celled tumour. Is locally destructive. Requires complete removal with a portion of surrounding bone.

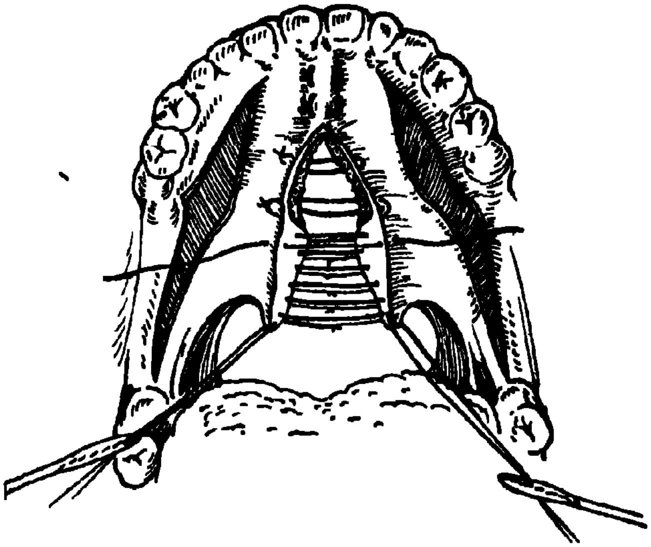


Fig. 139.—Cleft palate. Denis Browne's operation.

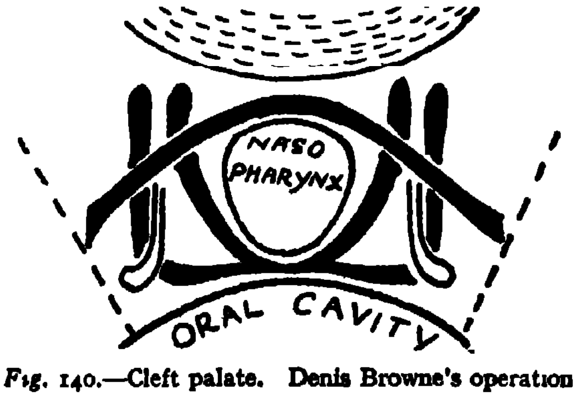


Fig. 140.—Cleft palate. Denis Browne's operation.

The Jaws, *continued.*

Epithelioma begins in the gums, or spreads from the cheek or tongue. It rapidly invades the underlying jaw.

Pyorrhœa Alveolaris (Riggs' disease).—A chronic suppuration between the roots of the teeth and the gums, with the infection slowly spreading through the periodontal membrane to the alveolus. Preceded and caused by a deposit of tartar. The pus forms pockets and sinuses beneath the gum margins. The gums retract from the teeth, and the teeth become loose. It may cause dyspepsia, a chronic sapræmia, and even chronic arthritis or synovitis

TREATMENT.—Scaling teeth, slitting up sinuses, long-continued daily irrigation with antiseptics Treatment by vaccine prepared from the infecting organism.

Necrosis of the Jaw.—

CAUSES —Dental caries and alveolar abscess—Compound fracture of the jaw—Injury and sepsis during teeth extraction—Phosphorus (rare nowadays)—Tertiary syphilis—Tubercle (rare)—Mercury poisoning—Osteomyelitis after exanthemata.

IN PHOSPHORUS JAW.—Rare to-day because of strict Home Office Regulation. The disease starts round a carious tooth socket in those who work in the fumes of yellow phosphorus Great swelling of chronic course results from a new periosteal bone formation. Sinuses open inside and outside the mouth There is great fœtor, with a tendency to sapræmia, dyspepsia, and septic pneumonia The sequestrum is grey and porous, and very long in separating There is strong evidence that this is really a tuberculous caries. The tubercle bacilli have often been demonstrated in the discharge, and the patients usually die of phthisis.

TREATMENT —This is conducted on general principles.

Dental Cysts and Tumours.—

1. SIMPLE DENTAL CYST —Found in the neighbourhood of the roots of carious teeth, generally the upper first molars and bicuspids Probably due to the irritation of an epithelial rudiment of the enamel organ. A painless swelling expands the bone, which presents egg-shell crackling. It contains mucoid material and some epithelial débris (*Fig.* 141)

   TREATMENT.—Removal of the tooth, with free opening of the cyst cavity, and mucosal flap.

2. FOLLICULAR ODONTOME, or dentigerous cyst of the jaw, is more common in the lower jaw It is caused by an expansion of the follicle which contains the developing permanent tooth. Hence it is always associated with the absence of one of the adult teeth The body of the jaw is expanded and thinned, the bone becoming like parchment. Inside the cavity the crown of the missing tooth is found (*Fig.* 142).

   TREATMENT.—By free opening and emptying, and mucosal flap.

3. EPITHELIAL ODONTOME, or fibrocystic disease of the jaw, usually affects the lower jaw, and in young people. It is caused by a proliferation of the epithelium of the tooth germ or enamel organ in the form of columns of cells resembling an epithelioma, but having a benign course and undergoing a cystic degeneration. A huge mass is formed, which expands the whole body of the jaw, the skeleton of which forms a bony framework for the cysts.

Treatment.—By removal of a large part of the jaw, usually nearly half the mandible.

4. FIBROUS ODONTOME.—An unerupted tooth surrounded by hard fibrous tissue.
   Treatment.—Complete removal of the tumour.
5. COMPOSITE ODONTOME.—A tumour consisting of enamel, dentine, and cement; often laminated. Extremely hard. May be large and invade the maxillary antrum and nasal cavity.
   Treatment.—Complete removal.
6. CEMENTOME —Extremely hard tumour growing from a tooth.
7. RADICULAR ODONTOME.—Occurs in connexion with the root of a particular tooth, seen in old people.

8 MYELOMA, when of central origin, may break down to form large blood-cysts. It also produces great expansion of the jaw.

9. CYSTS OF THE ANTRUM.—*See* p 328.

Innocent Tumours.—Fibroma, osteoma, cysts, and polypi.
Treatment —Local removal

Malignant Tumours.—Sarcoma or carcinoma. Sarcoma is much the commoner. It causes a steady enlargement of the jaw, and if the antrum is invaded there occurs the bulging of its five surfaces· (1) The cheek; (2) The orbital surface with exophthalmos; (3) The nasal surface, with nasal obstruction and epiphora, from blocking of the nasal duct; (4) The palate; (5) The zygomatic surface, producing swelling behind the jaw. Pain is very great and almost constant.
Treatment.—By radiation or by excision of the jaw.

Diseases of the Maxillary Antrum.—

SUPPURATION —

Causes.—Carious teeth, especially the molars and premolars. Suppuration in the nose or accessory nasal sinuses.

Symptoms —Pain and tenderness, with neuralgia of the infra-orbital nerve. Intermittent discharge of pus from the nose This may be seen to come from the middle meatus under the middle turbinate bone and above the inferior. It runs out when the patient holds his head forward, and trickles into the pharynx when he lies down. This is due to the fact that the opening into the nose is in the upper part of the antrum

Fig. 141.—Dental cyst, found at the root of a carious tooth.

Fig. 142 —Follicular odontome (dentigerous cyst). The crown of the unerupted tooth lies in the cavity of the cyst.

Diseases of the Maxillary Antrum—Suppuration, *continued.*

IN ACUTE CASES the nasal aperture is usually blocked. The symptoms are more severe, and may merge into those of osteomyelitis.

SIGNS:—

Pressure Signs.—(1) Nasal obstruction and blocking of the nasal duct; (2) Some exophthalmos; (3) Fullness of the palate, with irregularity of the teeth; (4) Swelling of the cheek.

Transillumination by an electric lamp in the mouth shows a darkness instead of the normal rosy colour under the eye.

Transirrigation.—A sharp hollow needle is thrust through the nasal wall of the antrum and fluid injected by a syringe. This flows out from the natural opening in the nose, and turbidity of the fluid indicates suppuration.

TREATMENT.—Drain: (1) Through inferior meatal wall, and wash out antrum; or (2) Through canine fossa.

MUCOCELE, or cystic distension.—

CAUSES.—A cystic distension of the glands of the mucous membrane, or the formation of a dental cyst

SIGNS are merely those of slow painless distension, with atrophy of the anterior bony wall, which gives egg-shell crackling.

TREATMENT.—As in the last case.

Leontiasis Ossea.—Begins in young adults with no known cause. Consists in exuberant spongy exostoses growing from the cranial and facial bones, especially from the maxillæ, mandible, and nasals.

Produces hideous deformity, severe neuralgia, displacement of the eyes, and lastly pressure on the brain.

TREATMENT.—Early removal of the masses as they appear.

Inability to open the Mouth.—

CAUSES.—(1) Ankylosis of the joint, resulting from sepsis, tubercle, or osteo-arthritis; (2) Cicatrical contraction of the soft parts round the joint after injuries or operations; (3) Spasm of tetanus; (4) Reflex spasm from carious teeth, especially an unerupted wisdom; (5) Inflammatory swellings inside or outside the joint, e g., mumps or tonsillitis; (6) Malignant tumours of the parotid or inside the mouth.

TREATMENT.—Only possible or desirable in the first two cases.

EXCISION OF THE CONDYLE is difficult, and often impracticable. An attempt may be made to interpose a vulcanite plate or some soft tissues between the bone surfaces to prevent bony union.

ESMARCH'S OPERATION.—Removal of a wedge-shaped piece of bone from the angle of the jaw on both sides, the apex of the wedge being at the alveolar border. The masseter and internal pterygoid muscles are sewn together in the gap so as to prevent bony union.

CHAPTER XXVIII

AFFECTIONS OF THE NOSE

Anatomy of the Nasal Cavity (*Figs.* 143, 144).—

The cavity of the nose is divided into two chambers, roughly triangular in shape, with apex upwards and base on the palate. It is divided down the centre by the septum, which is composed of an anterior cartilage, superior ethmoidal plate, and posterior vomer.

Each nasal cavity opens externally by the anterior nares and posteriorly into the pharynx by the posterior nares; each is divided partially into three meatuses by the inward projection of two bony scrolls or shelves—the middle and inferior turbinate bones.

The Superior Meatus is at the highest point in the nasal cavity. It contains the superior turbinate bone and the openings of the sphenoidal sinus and the posterior ethmoidal cells.

The Middle Meatus is between the middle and inferior turbinates. It contains the openings of the maxillary antrum and some of the ethmoid cells and of the fronto-nasal duct from the frontal sinus.

The Lower Meatus, below the inferior turbinate, contains the lower end of the lachrymal duct

Fracture of the Nasal Bones.—

Results from direct violence. One or both bones are broken near their free margin Or the cartilage is torn from the bones. The septum may be broken at the same time, or it may be the only injury. Surgical emphysema and epistaxis are common complications

TREATMENT.—Should be undertaken immediately to prevent deformity. The bones are replaced by external pressure, aided by padded forceps internally. A moulded guttapercha splint is fixed over the bridge of the nose by a bandage for a week.

Deviation of the Septum Nasi.—

CAUSES —External injury—Congenital defects.

SYMPTOMS.—Unilateral nasal obstruction.

VARIETIES.—

CARTILAGINOUS —Usually traumatic.

BONY.—Usually developmental.

TREATMENT —Submucous resection of the deviated septum. The mucous membrane is separated from the septum, first on one side and then on the other. The mucous flaps are retracted from the septum by the wings of a bivalve speculum. The septum is removed by a swivelled spokeshave or by punch forceps. The nasal cavities are packed, so as to press the two mucous flaps together.

Flattening of the Bridge of the Nose.—Destruction or impeded growth of the nasal septum.

Flattening of the Bridge of the Nose, ***continued.***

CAUSES.—Injury, syphilis (usually congenital), tuberculous rhinitis (very rare).

TREATMENT.—Subcutaneous grafting of bone. Cartilage grafts tend to twist after operation.

Nasal Obstruction.—

SYMPTOMS AND RESULTS.—Mouth breathing. Liability to colds and inflammation of throat and respiratory tract. Poor chest development. Stupid appearance.

INFLAMMATORY COMPLICATIONS.—Any of the cavities opening into the nose may have their outlet blocked—the accessory sinuses become blocked and inflamed—the Eustachian tube block causes deafness, etc.

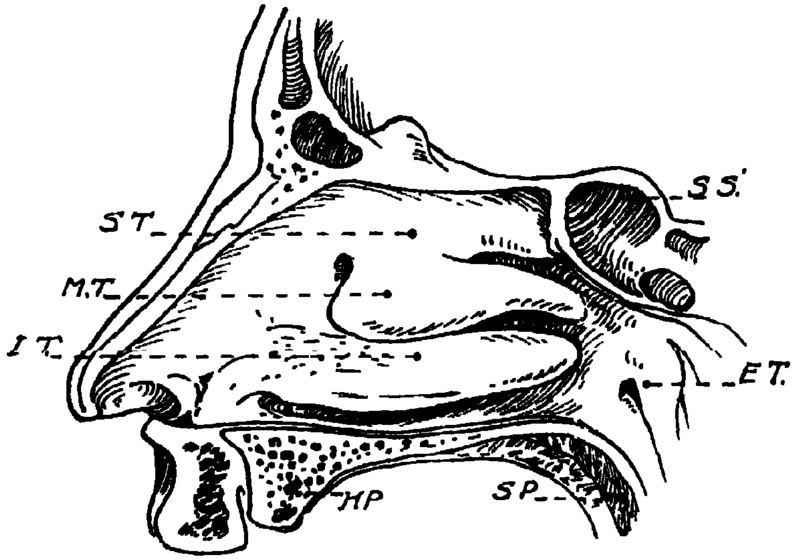


Fig 143.—Lateral wall of right nasal cavity. ***S.S***, Sphenoidal sinus, ***E.T*** Eustachian tube, ***S.P***, Soft palate; ***H P***, Hard palate, ***I.T.***, Inferior turbinate, ***M*** Middle turbinate, ***S.T.***, Superior turbinate.

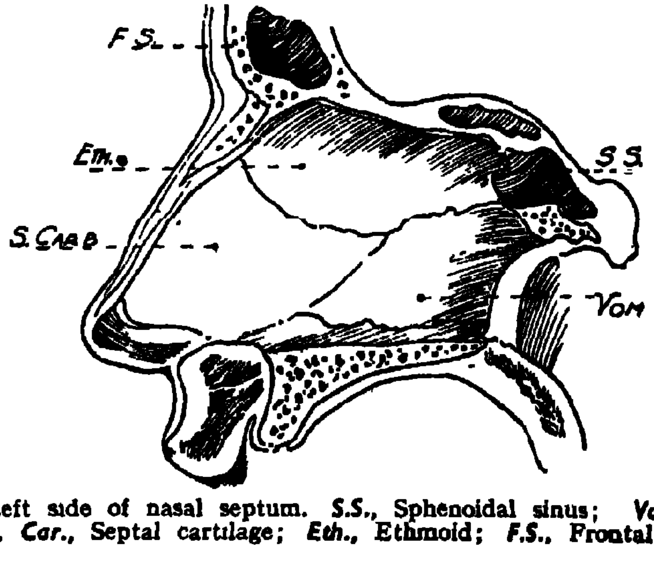


Fig. 144.—Left side of nasal septum. ***S.S.***, Sphenoidal sinus; ***Vom.***, Vomer; ***S. Car.***, Septal cartilage; ***Eth.***, Ethmoid; ***F.S.***, Frontal sinus.

CAUSES AND TYPES OF OBSTRUCTION.—

1. Swelling of the Mucous Membrane.—
   Rhinitis: simple, tuberculous, or syphilitic.
2. Obstruction Due to the Septum.—
   - *a* *Spurs* of cartilage and bone growing from the vomerine crest. Treat by cutting through the mucoperiosteum and sawing off the crest.
   - *b*. *Deviations* of the septum. These may be traumatic in origin, or come from ill-development Treat by submucous resection if there is no other primary cause of the obstruction.
   - *c*. *Hæmatoma* and abscess of the septum Treat by incision.
3. Diseases of the Ethmoid and other Accessory Sinuses.—*See* pp. 327, 332.
4. Diseases of the Turbinate Bones.—
   - *a*. *Vasomotor Engorgement*.—Much reduced by cocaine or adrenaline injections. Treat by: (i) Hygienic measures; (ii) Iron and strychnine internally; (iii) Cautery of the inferior turbinate.
   - *b*. *Nasal Cavities are Small* in proportion to the turbinates. Usually unsuited for treatment. Small parts of the front end of the inferior turbinate may be removed by scissors or punch forceps.
   - *c*. *Enlargement of the Middle Turbinate Bone* —Often associated with severe headache. Treat by removal by the scissors and forceps.
   - *d*. *Moriform Hypertrophy of the Posterior End of the Inferior Turbinate.* —Remove by a snare.
5. Blocking of the Cavity.—Foreign bodies, polypi, new growths, etc.
6. Blocking of the Posterior Nares —Adenoids, fibrosarcomatous polypi

Foreign Bodies in the Nose.—Beads, peas, etc., introduced by children, or rhinoliths—calculi formed by the deposit of calcareous salts in chronic rhinitis

SIGNS —Nasal obstruction, offensive purulent discharge.

TREATMENT.—Removal either by syringing or by forceps under an anæsthetic

Rhinitis.—

ACUTE —

Catarrhal —Ordinary 'cold in the head'.

Septic.—Associated with foreign bodies, septic wounds, and sinusitis.

Gonorrhœal —Spreads from the lachrymal sac.

Diphtheritic.—Spreads forward from the pharynx.

CHRONIC.—

Specific.—Lupus, spreads from the face or tear-duct. Syphilis, congenital (snuffles) or acquired.

Hypertrophic.—Causing considerable intermittent or permanent engorgement and hypertrophy of the nasal mucous membrane, chiefly over the inferior turbinate bones.

Signs.—Unilateral or bilateral nasal obstruction. Altered tone to the voice. The inferior turbinate bone appears enlarged, and encroaches upon the inferior meatus of the nose. This enlargement quickly subsides on syringing with cocaine or adrenaline.

Treatment.—Sprays, simple or astringent (e g., boracic acid or protargol, gr. j ad ℥ij). Touching with the electro-cautery. Removal of a part of turbinate bone.

Rhinitis, Chronic, *continued.*

Atrophic.—The mucous membrane is dry and shrivelled. The septum is often deflected. Pharyngitis and laryngitis are frequent accompaniments.

Nasal cavities are enlarged by an atrophy of their walls. Scabs and crusts cover the mucous surfaces *Ozæna* is the most characteristic sign A penetrating nauseous odour is caused, which the patient cannot smell. Sometimes associated with sinus disease.

Treatment.—Copious alkaline douches, followed by oily sprays of liquid paraffin, menthol, etc. Mechanical removal of crust and scabs, packing with gauze soaked in 5 per cent ichthyol in glycerin.

Accessory Sinusitis (*see also* Diseases of the Maxillary Antrum, p. 327).—The following sinuses open into the nose:—

Sphenoidal Posterior Ethmoidal Cells	Into the spheno-ethmoidal recess at the highest part of the nasal cavities.
Middle and Anterior Ethmoidal Cells Maxillary Antrum Frontal Sinus	Into the middle meatus or the hiatus semilunaris, under cover of the middle turbinate bone.

Nasal Duct.—Into the inferior meatus

CAUSES.—Bacterial invasion after catarrh, foreign bodies, operations, exanthemata.

SYMPTOMS AND SIGNS.—

Headache, which is either localized over the sinus, e g., over the frontal, or general, owing to the proximity to the base of the brain.

Neuralgia.—From pressure on nerves, e g, the supra-orbital by frontal disease

Purulent Discharge from the nose, which, except in the case of the sphenoidal and posterior ethmoidal sinuses, comes from the middle meatus.

Rhinoscopy.—A mass of polypi and granulation tissue occupies the place of the middle turbinate bone. Bare bone can often be felt. The swelling of the soft parts does not diminish much by cocaine or adrenaline injections

Radiography may show a shadow in a frontal or even in a sphenoidal sinus

Transillumination shows a shadow in the affected sinus, especially in the antral or frontal sinus.

DIFFERENTIAL DIAGNOSIS.—Antral and frontal sinusitis are recognized by the local tenderness, pain, and swelling, and by the evidence of transillumination

The sphenoidal sinus may give a shadow by the X rays if it is full of pus.

TREATMENT.—

For Frontal Sinusitis.—Removal of the anterior wall of the sinus, and draining into the nose.

For Ethmoidal and Sphenoidal Suppuration, an attempt may be made to get at the focus of disease through the nose, after clipping away the middle turbinate bone, or through the os planum of the ethmoid by an incision on the inner wall of the orbit. This is only necessary when there is an external fistula or when internal drainage and operation have failed. The sphenoidal sinus may also be reached in the midline of the nose by a submucous resection of the septum.

COMPLICATIONS OF SINUS DISEASE.—

ORBITAL.—Cellulitis or abscess in the orbit.

VISUAL.—From optic neuritis or mere septic absorption.

CEREBRAL.—Extradural or cerebral abscess, meningitis, cavernous sinus thrombosis.

MUCOCELE OF THE FRONTAL SINUS.—As in the case of the antrum, the frontal sinus may be distended by a mucous cyst. This causes expansion and thinning of its bony walls, which give an egg-shell crackling. A swelling appears on the forehead, and the eyeball becomes displaced downwards and outwards.

TREATMENT.—Removal of as much of the anterior bony wall as possible, and of all the mucous lining.

Nasal Polypi are of two different origins: (1) Inflammatory; and (2) Neoplastic.

1. MUCOUS POLYPI are merely œdematous granulations hanging from the surface of a diseased ethmoid bone which is affected by rarefying osteitis or caries. They occur in young adults, and cause nasal obstruction, often bilateral. Grow from the middle and superior turbinate bones. They frequently undergo cystic degeneration from the development of cysts in the glands of the mucous membrane covering them. After local removal they recur within a few months.

   TREATMENT.—(*a*) Removal by wire snare under cocaine; (*b*) Usually a thorough erasion of the lateral mass of the ethmoid or the opening of a suppurating sinus is required, so as to remove the diseased bone.

2. FIBROUS AND SARCOMATOUS POLYPI.—Consist in all gradations between fibromata and sarcomata, usually beginning as the former and ending as the latter. Occur in children and adolescents most commonly. Grow from the base of the skull and occupy the nasopharynx. Cause nasal obstruction, with sanious discharge.

   PRESSURE SIGNS.—The tumour may: (1) Push down the velum palati and cause asphyxia; (2) Expand the nasal cavities and produce a widening of the nasal bridge; (3) Press the eyeballs outwards; (4) Extend into the base of the brain.

   IF TUMOUR IS MALIGNANT, secondary growth may occur in the lymph-glands of the neck. In these cases the primary growth is probably a lymphosarcoma of the pharyngeal tonsil.

   TREATMENT is possible only in the early stages.

   1. Removal by a snare through the anterior nares.
   2. Removal through the mouth after splitting the soft palate.
   3. Removal from the face after turning up the soft parts and enlarging the anterior nares by temporary displacement of the maxillæ.

   Moure's operation of lateral rhinotomy has been given up in favour of removal of the neoplasm with electrosurgery and packing 1-mg. radium needles into the cavity containing the neoplasm for 3–4 days.

Adenoids.—A hyperplasia of the lymphadenoid tissue at the back and roof of the nasopharynx.

CAUSES.—Constitutional delicacy, which causes repeated nasal catarrh. Hygienic defects, e.g., insufficient ventilation. Want of exercise and a consequent slovenly method of breathing.

Adenoids, *continued.*

SIGNS AND SYMPTOMS.—A foliate and more or less symmetrical lymphoid mass fills up the nasopharynx. This can be seen by posterior rhinoscopy and felt by the finger-tip The size varies from day to day, becoming much larger if the child has a cold Mouth-breathing. The nasal passages are obstructed and the mouth kept habitually open. It becomes hard and parched. Snoring is marked at night. Discharge of sero-pus from the nostrils and nasopharynx.

RESULTS AND SEQUELÆ.—

ON NOSE.—The nostrils become narrow and slit-like, from a falling in of the lateral cartilages. The vomer and septal cartilage become distorted, because the nasal cavities are not large enough for their growth

ON MOUTH.—The palate is high, narrow, and arched Teeth arranged irregularly, being crowded together from insufficient palate growth. Most noticeable after seven years, when the large permanent teeth are making their appearance.

ON EARS.—The nasopharyngeal catarrh affects the Eustachian tubes, and causes chronic obstruction of these, with deafness, and frequent otitis media and its complications It is by far the commonest cause of deafness.

ON TRUNK.—The chest, from blocking of the air inlet through the nose, becomes either flat or pigeon-breasted There is great liability to bronchitis and phthisis Kyphosis of the spine accompanies this change.

ON THE MIND.—Mental torpor and stupidity occur from defective aeration of the nerve centres, and from the restless and bad sleep produced by the nasal obstruction. The gaping mouth, deafness, dribbling, all increase the appearance of feeble-mindedness.

TREATMENT.—

CLIMATE —An equable, dry climate like that of Egypt will often cure a slight case.

EXERCISES.—Systematic 'breathing exercises' in conjunction with the application of 2 per cent argyrol drops locally will cure most cases of adenoids if persisted in for long enough, but in the majority of cases this is quite impracticable.

OPERATION.—Removal by a curette under a general anæsthetic; careful training in nose-breathing is very important afterwards.

Epistaxis.—

CAUSES.—Traumatism. After operations. Fractured skull, etc. Ulcer on the septum. Vasomotor disturbances: (*a*) In adolescents, (*b*) In adults with high blood-pressure. Certain blood conditions, e g , hæmophilia.

TREATMENT—after examination to discover the cause.—

1. Cauterize the bleeding spot if possible.
2. Plug the nasal cavity with gauze soaked in adrenaline.
3. Plugging the nares (anterior and posterior) is seldom required.
4. An inflating plug introduced into the nose and then inflated.
5. Calcium chloride or lactate in drachm doses by the mouth.

CHAPTER XXIX

AFFECTIONS OF THE TONGUE

Congenital Malformations.—

ABSENCE or ARRESTED DEVELOPMENT (very rare).

BIFID or SPLIT TONGUE may require suture for appearance sake.

TONGUE-TIE, or ANKYLOGLOSSIA.—The frænum is too short, and so sucking and speaking are difficult. It is very rare, though often alleged. Careless division in spurious cases has often caused severe bleeding, septic ulcers, or dangerous tongue-swallowing

TONGUE-SWALLOWING.—Excessive mobility allows the tongue to fall back, and may cause death by suffocation.

Hypertrophy of the Tongue.—

1. MACROGLOSSIA is a condition of lymphangioma which is usually congenital, but sometimes acquired Clear vesicles are seen on the surface, usually near the tip, and the whole tongue enlarges. Lymphatic cysts are formed beneath the mucous membrane, the papillæ hypertrophy, and there are also newly-formed blood-vessels and small-round-celled infiltration. The organ is liable to recurrent attacks of glossitis, after each of which it is larger than before It becomes so large that it cannot be retained in the mouth, and secondary changes take place in the lower jaw. TREATMENT.—By V-shaped excision.
2. SIMPLE MUSCULAR HYPERTROPHY may occur in idiots, or without any other lesion.
3. INFLAMMATORY HYPERTROPHY may be seen after any variety of glossitis, but most frequently after syphilitic.
4. IN MENTAL DEFECTIVES.
5. IN ENDOCRINE DISORDERS, e.g., myxœdema.

Injuries of the Tongue.—

Wounds are most commonly produced by the teeth, either in epilepsy or eclampsia, or by blows on the jaw when the tongue is protruding. Hæmorrhage is severe, but healing is rapid.

PUNCTURED wounds, as by pipe stems, may have a foreign body in their depth, and this may give rise to severe secondary hæmorrhage, or to chronic inflammatory thickening.

Acute Parenchymatous Glossitis occurs generally in adult men following stings of insects and penetrating wounds with infection. Cold, mercurialism, and debility are predisposing causes

Rapid painful swelling takes place, which makes speech and swallowing, or shutting the mouth, impossible.

Death may occur from asphyxia or sepsis, but recovery is the rule, with some superficial sloughing.

TREATMENT.—In bad cases by free longitudinal incisions. Mild cases—heat to neck, and hot mouth-washes, with ice to suck in the intervening periods.

Affections of the Tongue, ***continued.***

Streptococcal Glossitis begins in the neighbourhood of the submaxillary gland, and ends as a rapid cellulitis of the neck, with œdema of the glottis.

TREATMENT.—By chemotherapy and tracheotomy, if necessary.

Staphylococcal Glossitis usually results from wounds and injuries. An indurated swelling forms, which develops into an abscess deep in the tongue substance

TREATMENT.—Free opening in the mouth, or, in deep cases, through the mid-line of the neck above the hyoid followed by sulphathiazole therapy, intramuscularly or intravenously.

Mercurial Stomatitis is accompanied by bad breath, spongy gums, and superficial sloughing

TREATMENT.—By chlorate of potash, with astringent gargles.

Gangrenous Glossitis may result from acute glossitis, or as an extension from cancrum oris. In the former case natural cure by the separation of the slough is the rule

Acute Superficial Glossitis occurs in many septic mouth conditions and in dyspepsia and gout.

SIMPLE OR DYSPEPTIC —The tongue becomes indented by the teeth, and in bad dyspeptic cases it is raw and excoriated.

TREATMENT.—By chlorate of potash, and that for the dyspepsia.

HEMIGLOSSITIS is rare, and usually of nervous origin and accompanied by herpes.

APHTHOUS GLOSSITIS, OR THRUSH, occurs in infants from the growth of the *Oidium albicans* The tongue and mouth become red, and then are covered with white patches, which consist of masses of the organism. It is very contagious among children

TREATMENT.—By care of milk supply, and cleaning the mouth with alkaline and boracic washes.

ERYTHEMA MIGRANS, or wandering rash, occurs rarely and in debilitated children. Red, smooth patches are seen, surrounded by a raised yellow border The latter spreads like ringworm, and neighbouring rings may intersect It causes itching and salivation, and is very chronic. No special treatment is available.

HERPES is most common in neurotic women. It is very painful, leaves troublesome ulcers, and is prone to repeated recurrences.

TREATMENT.—By application of cocaine or of carbolic acid.

VINCENT'S STOMATITIS (Trench Mouth).—Infection by Vincent's spirillum, in young adults Mouth covered in grey patches surrounded by a red zone.

TREATMENT.—Ascorbic acid (50 mg. t.d.s.) and potassium chlorate mouth-washes. Resistant cases may need treatment by painting the area with 10 per cent novarsenobenzol or Milton.

Chronic Superficial Glossitis.—

LEUCOPLAKIA OR LEUCOKERATOSIS.—

AETIOLOGY.—Only occurs between twenty and sixty, and very rarely in women. Syphilis, smoking to excess, especially rough pipes, taking raw spirits and hot foods, gout, rheumatism, and dyspepsia, are all predisposing causes.

PATHOLOGY.—Papillæ disappear. A layer of small round cells appears between the Malpighian layer and epidermis, and latter becomes horny. Pathological types:—

1. *Swelling of Papillæ.*
2. *Leucoplakia.*—Overgrowth and cornification of epithelium.
3. *Raw Glazed Tongue.*—Epithelium is shed, leaving raw tongue without any papillæ.
4. *Cracks and Fissures*—Due to contraction of fibrous tissue, leaving fissures in furrows. This is the pre-carcinomatous stage
5. *Epithelioma.*—Frequently follows.

SYMPTOMS.—The tongue feels hard and dry, and thirst is prominent. Tenderness and rawness are felt, especially when the patch is denuded of its covering, when ulcers, warts, and fissures are apt to form.

COURSE.—Lasts many years and is practically incurable. Various stages of the disease may co-exist. It frequently gives rise to epithelioma.

TREATMENT.—Avoiding smoking, and sweet, sour, sharp, or strong articles of diet. Alkaline gargles, borax and glycerin, and also boracic or eucalyptus ointment, put on at night after carefully drying the tongue. Also chromic acid, gr. v ad ℥j. Excision of limited patches, warts, ulcers, or fissures. In resistant cases, Butlin's operation, which consists of removing the superficial affected layers of the tongue, is of value.

SMOKER'S PATCH—Localized area of leucoplakia from which the heaped-up epithelium has been shed.

HYPERKERATOSIS (Black Tongue)—Patch of epithelium in the midline in front of the circumvallate papillæ becomes dark or black, and the papillæ enlarge to form long waving hairs. The colour is due to micro-organisms No special treatment is required

Simple Ulcers occur in superficial glossitis, and are very intractable when they become chronic.

PAINT with chromic acid, and EXCISE if they do not heal at once, lest cancer

Herpetic Ulcers occur in children or young adults as an acute condition, with some malaise, as a vesicular eruption, followed by ulceration.

Chlorate of potash is the best TREATMENT.

Irritation Ulcers affect edges of the tongue adjacent to broken carious teeth or badly-fitting tooth-plates Ragged and sloughy base and edges. No induration at base. Rapidly heal when tooth is removed. If left may be the starting-point of epithelioma.

Dyspeptic Ulcers.—Generally on the dorsum near the tip. Associated with a severe grade of dyspepsia. Shallow and indolent. No induration. Painful.

TREAT the dyspepsia, and paint ulcer with lunar caustic.

Mercurial Ulcers occur after the separation of superficial sloughs, with all the fœtor and other signs of stomatitis.

Tuberculous Ulcers.—Complicate phthisis or lupus. Especially near tip, but may occur anywhere. Left by the breaking of an abscess. Shallow ulcer with sharp undermined edges and no induration. The surface is pale and flabby. Very painful.

TREATMENT.—By excision under cocaine.

Affections of the Tongue, ***continued.***

Syphilis of the Tongue.—

PRIMARY.—As in lip chancre. Much matting and enlargement of glands. Much infiltration of the base, but not much induration.

SECONDARY.—Shallow, multiple, painful, small ulcers, especially round the edges and under surface. Mucous patches and condyloma (Hutchinson's wart).

TREATMENT.—Paint with chromic acid, gr. x ad ℥j, over ulcers. Specific treatment is also given and general mouth hygiene.

TERTIARY.—

1. CHRONIC SUPERFICIAL GLOSSITIS.
2. DIFFUSE FIBROUS THICKENING of the tongue. Chronic interstitial glossitis, generally combined with (1). Tongue large and hard. Distorted by furrows and fissures. Caused by fibrous tissue drawing in the surface in folds and furrows.
3. GUMMA WITH ULCERATION.—Patient is generally about forty. Begins as a swelling in the tongue substance. In mid-line, far back, is common situation, or at the side. Breaks and discharges 'gummy material'. Quite painless ulcer is left. Base formed by tough slough (like wet wash-leather). Sharply-cut edges—'punched-out'. No induration of the base. No fixation of the tongue. No enlargement of glands. Remains stationary a long time. Scar may give rise to epithelioma.

Simple Tumours of the Tongue and Floor of the Mouth.—

SALIVARY GLAND TUMOURS may grow from the submaxillary, sublingual, or incisive glands. The majority are endotheliomata, and consist of endothelial columns and cartilage.

RANULA is an obstructive or retention cyst, usually of the sublingual, rarely of the incisive glands (*see* p. 344).

SALIVARY CALCULUS, with surrounding inflammation, may be impacted in the submaxillary duct, and form a hard swelling.

DERMOID CYSTS are usually in the mid-line in the floor of the mouth, between the mylohyoid and geniohyoglossi. These are sequestration dermoids. They may be lateral near the angle of the jaw, in which case they are probably tubulo-dermoids from a branchial cleft. They form a firm, elastic, yellow swelling, which projects below the chin.

TREATMENT.—By excision through the mouth in most cases.

PARASITIC CYSTS.—Cysticercus and hydatid cysts extremely rare.

LINGUAL TONSIL may be chronically enlarged or subject to a follicular abscess. In either case surgical treatment, excision or incision, will be required.

THYROGLOSSAL DUCT TUMOURS AND CYSTS.—These occur in two situations: (1) At the back of the tongue in the foramen cæcum, as a tumour or a cyst, which is red, vascular, and about the size of a pea or cherry, and has the structure of a thyroid adenoma; (2) In the substance of the tongue near the hyoid bone, in which case they form cysts lined by ciliated epithelium, and containing mucus.

BLOOD-CYSTS of the tongue are due to hæmorrhage taking place into these.

TREATMENT.—Excision.

LIPOMA may be: (1) Congenital, when it is large and deep-seated; (2) Superficial and small, occurring in old people, and becoming polypoid; (3) Deep-seated, occurring in late life, between the tongue and floor of the mouth.

FIBROMA.—Either polypoid or deep-seated. Is painless and of very slow growth.

ANGIOMA.—This may be arteriovenous, capillary, or cavernous, the last being the largest and most important Usually congenital.
EXCISION will be required to avoid ulceration and hæmorrhage.

PAPILLOMA occurs in children and adults as a simple new growth, and also in cases of superficial glossitis, when it is a precursor of cancer. It should always be removed.

Sarcoma of the Tongue is a very rare disease. It occurs in young adults as an elastic mass in the tongue substance Its nature is seldom recognized until removal has been done. In some cases it is encapsuled and comparatively benign, but in others (lymphosarcoma), which begin in the lingual tonsil, malignancy is well marked.

EPITHELIOMA OF THE TONGUE

Aetiology.—Much commoner in men, i e, about 85 per cent. Occurs generally between forty and sixty. Caused by rough pipe, much smoking, irritation ulcer, syphilitic scars, etc., and also the injudicious use of caustics.

BEGINS as —

1 WARTY OUTGROWTH, often in chronic glossitis
2 AN ULCER at edge, at junction of middle and posterior third Probably from a rough tooth. On the dorsum in chronic superficial glossitis.
3. CRACK OR FISSURE, especially in chronic glossitis.
4. SUBMUCOUS INFILTRATION (very rare).
5 EXTENSION from tonsils or larynx

Signs and Symptoms.—Usually begins on the anterior two-thirds, and towards the edges. Crateriform ulcer, or a warty growth Hard, everted (rolled-over) margins Sides slope down to a ragged, foul base. Bleeds on touching. Profuse fœtid discharge and profuse salivation Base of the ulcer is very indurated. Glands in the neck hard and enlarged due to secondary deposits and associated extension from secondary infection of the growth.

LATER.—Pain is very marked; often localized in the ear. Saliva constantly dribbles from the mouth. Tongue cannot be protruded owing to the involvement of intrinsic muscles Glands in the neck form a huge mass.

Extension.—Carcinoma of anterior two-thirds tends to invade the floor of the mouth, in the posterior third it extends on to the soft palate and pharynx.

Secondary Deposits.—

GLANDULAR.—The lymphatic glands of the neck are invaded generally within the first six months of the disease, and often before that. The submental and submaxillary groups are involved first, with growths at the tip or sides of the tongue; and the carotid group, extending from the skull to the sternum, is affected later. The most constant position is at

Epithelioma of the Tongue—Secondary Deposits—Glandular, ***continued.***

the carotid bifurcation. Usually in the early stages only the glands of the same side as the growth are affected, but later those of the opposite side become involved (*Fig.* 145).

VISCERAL.—The liver, lungs, heart, suprarenals, and other viscera are affected with extreme rarity.

Complications.—

GLANDS IN NECK.—Form large cystic swellings. Break and form foul septic ulcers. Cause death by tracheal pressure, or by pressure dysphagia.

SEPTIC PNEUMONIA

SECONDARY HÆMORRHAGE.

Course.—Patient generally dies within one year.

Diagnosis.—

From OTHER ULCERS.—*See* p 337.

From ACTINOMYCOSIS.—Pus, with characteristic granules.

From INFLAMMATION ROUND SALIVARY CALCULUS.—

Although there may be great induration, there is no real fixation.

An incision will reveal the stone

Every ulcer, wart, or fissure which does not at once yield to local or specific treatment should be removed and examined for epithelioma.

Treatment.—

RADIUM —*The application of radium is now the method of choice* Needles of radium (0·5 mg., 1 mg , or 2 mg) are buried in the substance of the tongue, in and around the growth Each tube is tied in by attached threads and stitched in. The radium tubes are left in place for about seven days. The neck is afterwards surrounded by a moulded collar in which radium needles are inserted so as to lie as closely as possible to the lymph-gland areas. Growths in the posterior third are best treated by teleradium.

ADVANTAGES OF RADIUM OVER OPERATION.—Much less painful. Less mutilating Less dangerous Applicable to situations when excision is impossible (e g., far back, involvement of fauces or floor of mouth).

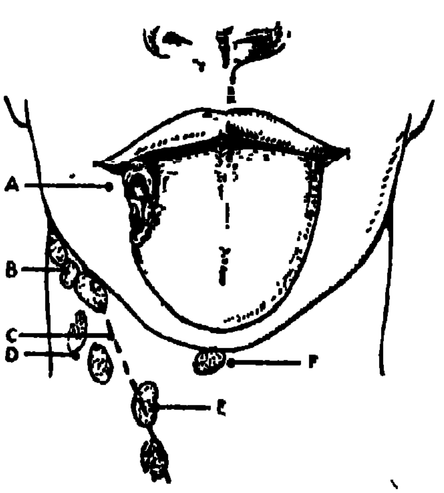


Fig. 145.—Diagram of epithelioma of tongue and glands. A, Growth starting at lateral border of tongue, B, Glands at angle of jaw, C, Line of sternomastoid muscle; D, Carotid glands, upper set, E, Carotid glands, lower set; F, Submental glands.

OPERATIONS FOR CANCER OF TONGUE.—

Indications.—When radium is not available. When the cancer is on the tip or side of tongue, making excision of the anterior part of half the tongue easy (a matter of opinion). When the lymph-glands are palpably enlarged, it is wiser to do a block dissection than to trust to a radium collar (a matter of opinion.)

Preliminary to all operations, even insertion of radium needles—Remove carious teeth or stumps. Scale and clean the remaining teeth. Rub off all fur possible from the tongue with dilute antiseptics. Treat any dyspepsia. Spray mouth with 1–80 carbolic, or 1–2000 biniodide of mercury, or protargol and glycerin 1 per cent solution

In Bad Cases, i.e., where operation is a very extensive one, where there is a foul fungating mass, a preliminary injection of streptococcal serum may be given as a prophylactic.

Methods of Operation.—

Half the Tongue in its anterior two-thirds may be removed through the mouth, if the tongue is freely movable and the disease does not near the midline (Whitehead's operation).

The Whole Tongue may be removed by (1) An incision under the jaw from ear to chin (Kocher), (2) Splitting the lower lip and jaw through a median incision (Symes).

Diathermy —The use of the diathermy cutting knife should be employed for division of the lingual tissues. It destroys cancer cells which lie in or adjacent to its line of action There is much less after-pain and much less hæmorrhage.

IN REMOVAL OF THE WHOLE TONGUE BY ANY METHOD: PRECAUTIONS —

Preliminary Tracheotomy —At the time of operation, or a few days previously Use Hahn's tube surrounded by sponge, or a dilatable jacketed tube, or plug pharynx with a sponge

Or **Laryngotomy** at the time Easier—Quicker—Heals more rapidly afterwards.

Or **Intratracheal Anæsthesia** —The anæsthetic is given by a catheter passed down through the larynx. The constant stream of outgoing air prevents blood or other fluid from entering the trachea.

Secure the Stump by a Thread fixed to the cheek to prevent asphyxia.

TREATMENT OF THE GLANDS —Essential—usually performed 14 days after operation or radium therapy. Classified as follows.—

1. **Glands not Palpable.**—Deep X-ray therapy, teleradium, or block dissection
2. **Glands Palpable but Operable.**—Block dissection, followed by deep X-ray therapy
3. **Glands Palpable but Inoperable** —Interstitial irradiation and external irradiation.

AFTER-TREATMENT.—Leave the tracheotomy tube in place two to seven days in bad cases. Mouth-washes with Condy or 1–80 carbolic. Feed by rectum for twenty-four hours, by œsophageal tube for six days.

CAUSES OF DEATH AFTER OPERATION.—Shock—Recurrent or secondary hæmorrhage—Asphyxia from falling back of the stump on the larynx—Septic pneumonia—Septicæmia.

Epithelioma of the Tongue—Treatment, ***continued.***

OPERATION RESULTS.—Immediate mortality: intrabuccal operation, 7 per cent; extrabuccal operations, over 20 per cent. Cases alive at the end of three years without recurrence, 20 per cent.

RECURRENCE usually takes place in the stump within six months, or in the cervical glands at a later period

Palliative Treatment.—Removal of carious teeth, and keeping the mouth clean with antiseptics, e g., hydrarg bicyanide 1–1000

FOR PAIN —Insufflation with powder containing orthoform. Packing ulcers with iodoform gauze. Excision of lingual nerve. Lastly, morphia by hypodermic injection.

FOR SALIVATION AND FŒTOR.—Atropine as an injection, and iodoform locally.

CHAPTER XXX

AFFECTIONS OF SALIVARY GLANDS, TONSILS, PHARYNX, AND ŒSOPHAGUS

AFFECTIONS OF THE SALIVARY GLANDS

Parotitis.—

VARIETIES —(1) Acute, (2) Chronic

1. ACUTE —

a. NON-SUPPURATIVE.—Epidemic parotitis or mumps: Infectious, and affects usually children; both glands are generally affected by a parenchymatous inflammation which never suppurates. Often coincident (or metastatic) inflammation of the genital organs. testis, breast, or ovary Occurs in this order of frequency.

Treatment —Medical

b. SUPPURATIVE.—Usually an ascending duct infection from mouth, but may be pyæmic, a blood infection from any distant septic focus. Duct infection occurs with the dry septic mouth in patients following operation, and during fevers Also because of the diminished salivary flow from the absence of food stimulus.

Treatment.—Fomentations, and early incisions parallel to the zygoma so as to avoid injury to the facial nerve

2. CHRONIC.—(*a*) Simple. (*b*) Actinomycosis. (*c*) Tuberculosis. (*d*) Syphilis. The last three are very rare.

AETIOLOGY —Caused by infection from the mouth

SYMPTOMS —Painful swelling of the gland.

TREATMENT —Massage, or injection of lipiodol into the duct. Inflammation of the other salivary glands is rare, and calls for no comment.

Salivary Calculus is common in submaxillary duct, but rare in parotid. It forms a hard swelling in the floor of the mouth, and may lead to salivary obstruction or fistula. It can be seen by X rays. It has been mistaken for epithelioma.

TREATMENT —Excision through the mucous membrane

Salivary Obstruction may be due to: (1) Calculus: (2) Cicatrization: or (3) Injury Rare, except in the submaxillary gland.

SYMPTOMS.—Painful swelling of the gland after meals.

TREATMENT.—By removal of the obstruction, or of the gland.

Sialography.—One to two c.c. of lipiodol are injected into parotid duct. An X-ray will then show deformities or dilatations of the ducts or acini.

Salivary Fistula.—Is rare, except in parotid duct. Caused by operation wounds and injuries.

TREATMENT by passing sutures, or drainage tubes, from the intrabuccal opening to the fistula.

Affections of the Salivary Glands, ***continued.***

Tumours of the Salivary Glands.—

RETENTION CYST or RANULA occurs in the floor of the mouth. Forms a bluish swelling in the floor of the mouth, and is always unilateral. Submaxillary duct overlies it. It is a myxomatous degeneration of a mucous gland on the floor of the mouth, and it may be sublingual, or a solitary mucous gland, e.g., gland of Blandin and Nuhn.

TREATMENT.—Excision.

SIMPLE TUMOURS.—These, as well as malignant growths, occur commonly in the parotid, and rarely in the submaxillary glands. Formerly regarded as mixed tumours, but now are stated to be true adenomata with areas of mucoid degeneration, the cells of these areas staining to give the appearance of cartilage.

SIGNS.—A firm elastic or cystic swelling, which moves freely, and is usually limited by the zygoma, anterior margin of the masseter, sternomastoid, and angle of the jaw.

SYMPTOMS —Usually painless, but unsightly. Quite slow in growth, but may enlarge suddenly and become malignant.

TREATMENT —Excision

If doubt exists as to complete excision, 1 mg of radium inserted into the growth for 3–4 days will prevent any recurrence.

VON MIKULICZ' DISEASE —Chronic symmetrical enlargement of all the salivary glands, and including the lachrymal glands The pathology is unknown It is probably a chronic inflammation, and not a new growth.

TREATMENT.—X-ray therapy. Removal of sublingual and submaxillary glands.

MALIGNANT TUMOURS —These are usually adenomata which have become malignant, but primary sarcoma or carcinoma may also occur.

SIGNS —Fixation of the tumour to the skin and underlying tissues. Facial paralysis from involvement of the facial nerve Extension into the zygomatic fossa and encroachment upon the mouth and pharynx

TREATMENT.—Excision, but this is only possible if the case is seen early.

AFFECTIONS OF THE TONSILS

Acute Tonsillitis.—According to the part involved this may be: (1) Follicular, (2) Parenchymatous; (3) Superficial. But this distinction is probably quite artificial and unnecessary.

FOLLICULAR TONSILLITIS is the usual form.

CAUSES.—Usually due to a *Streptococcus pyogenes.* Contact with septic wounds (hospital throat), scarlet fever, rheumatism, chronic enlargement of the tonsils. May occur in tonsillar remains.

SYMPTOMS —Swallowing is very painful, the glands enlarge under the jaw, and the temperature rises to 103°–105° F.

SIGNS.—Both tonsils become swollen and inflamed, and all their lacunæ filled with mucopus, which may also form a membrane over the surface.

DIAGNOSIS must be made from scarlet fever by absence of rash.

TREATMENT.—Administer purge at commencement of illness. Chemotherapy. Local applications of eucaine and the use of aspirin gargles.

Peritonsillitis or Quinsy.—Suppuration in or around the tonsil, usually the latter, occurring between the tonsil and its bed.

SIGNS.—Bright red swelling of the fauces and soft palate, usually much more marked one side than the other. The tonsil itself is often buried in the swollen faucial pillars. Inability to swallow is almost absolute, and the pain and constitutional disturbance are severe. If left alone, about eight days elapse before the abscess bursts, which it generally does through the soft palate or anterior faucial pillar. Rapid recovery follows. Fluctuation and pointing of the abscess can be seen and felt with difficulty, because the patient cannot open the mouth.

TREATMENT.—an incision directly a soft spot can be found. This generally has to be made through the soft palate, and runs parallel to the anterior faucial pillar.

Chronic Tonsillitis. Hypertrophy.—

CAUSES —Sepsis, cold, debility, insufficient exercise. A previous attack of acute tonsillitis makes recurrent attacks the rule, especially in adults, and after each of these the tonsils are larger than before.

STRUCTURE AND ASSOCIATED CONDITIONS.—The enlarged tonsils may: (1) Stand out as protuberant masses which almost touch one another; or, (2) They may lie deeply concealed within the faucial pillars Above the tonsil and between the faucial pillars is a recess, the supratonsillar fossa, into which the upper follicles open. The large follicles show open mouths, or these may be plugged with bacterial and mucoid débris. A lobe runs down on to the lateral surface of the tongue. In children the pharyngeal tonsil is generally also enlarged (*see* ADENOIDS, p. 333).

SYMPTOMS.—Thickness of speech. Mouth-breathing is common because of the adenoids. Liability to repeated acute attacks of tonsillitis is the most serious result. Also considerable debility may result from the chronic septic absorption.

TREATMENT —*Operative removal* is the only satisfactory treatment.

Removal by the Guillotine should only be done in young children, and in them only for cases in which the tonsils project beyond the faucial pillars

Removal by Dissection should be regarded as the ideal method. It is the only method which gives a reasonable security for non-recurrence; the only method by which the whole tonsil and nothing but the tonsil can be removed; the best method for securing hæmostasis.

Essential steps: anæsthetic through nostril; head down position; efficient gag which gives full view; dissection after freeing the anterior faucial pillar. Hæmostasis by ligature, suture, or diathermy.

Syphilis of the Tonsil may be: (1) Primary (very rare); (2) Secondary superficial ulcers, which are common; (3) Gummata, or late tertiary ulceration, which cause great scarring and deformity.

New Growths of the Tonsil.—

VARIETIES.—

EPITHELIOMA, which usually begins in the fauces or tongue.

LYMPHOSARCOMA occurs at any age, but usually in young people, and is intensely malignant.

MIXED-CELL SARCOMA is rare.

SIGNS AND SYMPTOMS.—Rapid enlargement without pain or inflammation. In the case of epithelioma, deep ulceration soon occurs. In that of a sarcoma the mass is elastic, soft, and encapsuled at first. In

New Growths of the Tonsil—Signs and Symptoms, ***continued.***

all, the submaxillary cervical lymph-glands rapidly become enlarged, Dysphagia, asphyxia, or secondary hæmorrhage are the usual causes, of death, which occurs within one year.

TREATMENT —

RADIUM NEEDLES, buried in and around the growth. Good results from teleradium.

REMOVAL through the neck with all the lymph-glands of that side that can be found The incision is that of Kocher's tongue incision, and the external carotid is tied.

AFFECTIONS OF THE PHARYNX

Acute Pharyngitis is caused by septic absorption, and often accompanies scarlet fever, septicæmia, rheumatism, and syphilis Pain, swelling, and redness of the fauces and mucous membrane, with dysphagia. In septic cases, œdema of the glottis may occur and cause asphyxia.

TREATMENT —Inhalation of steam impregnated with carbolic or balsamic vapours. Salicylates by mouth. Multiple incisions in cases threatened by asphyxia.

Chronic Pharyngitis is caused by smoking, drinking raw spirits, over-use of the voice, e g., in clergymen and hawkers The vessels become atonic and dilated, and nodules of lymphoid tissue become prominent An excess of sticky mucus is secreted, which is tenacious and causes hawking and retching.

IN THE FOLLICULAR VARIETY the enlarged lymphoid nodules are prominent.

IN THE ATROPHIC VARIETY the mucous membrane is smooth and glazed, and may be covered by crusts.

TREATMENT —

AVOIDANCE of speaking, smoking, and drinking alcohol

SPRAYS of tannic acid, perchloride of iron, or protargol, in glycerin, or oily sprays containing menthol, eucalyptus, etc.

GALVANO-CAUTERY POINT to all the large lymph-follicles.

Post-pharyngeal Abscess.—

ACUTE.—Due to septic infection through abrasions, e g., swallowing a fish bone. Caused by pyogenic organisms, and develops between the pharynx and the prevertebral fascia. May cause asphyxia by suddenly bursting into the larynx Forms a tender, fluctuating swelling at the back of the throat.

TREATMENT —Incision through the pharyngeal wall, care being taken that the pus does not enter the larynx

CHRONIC.—Due to tuberculous disease of the upper cervical vertebræ. It forms with little pain or inflammation, deep to the prevertebral fascia, behind which it tracks out into the neck.

TREATMENT.—Open by an incision behind sternomastoid, before bursting or septic infection through the mouth has occurred.

Syphilis of the Pharynx.—(1) Secondary, in the form of superficial mucous patches, or snail-track ulcers; (2) Tertiary, in the form of submucous gummata and deep ulceration, which in the process of healing causes great deformity by tying down the soft palate to the pharyngeal wall.

Stenosis of the Pharynx.—Due either to cicatrization after scalds, or most often after syphilis. In the commonest type, the remains of the soft palate are so bound down to the posterior pharyngeal wall that the nasopharynx is shut off from the mouth. In the more serious type, fauces, soft palate, and posterior walls are contracted so as to constrict the passage from mouth to œsophagus.

TREATMENT.—Division of the stricture and passage of bougies If this fails, œsophagostomy will be required in severe cases.

Epithelioma is the only primary growth of the pharynx, and as a primary growth is rare. Its appearance, course, and treatment are similar to those of the disease at the back of the tongue.

Pharyngeal Pouch or Propulsion Diverticulum is a hernial protrusion of mucous membrane between the transverse and oblique portions of the inferior constrictor of the pharynx Is usually found in adults about thirty. Lies usually on the left side. Causes dysphagia by pressure.

DIAGNOSIS—Is facilitated by radiography after swallowing barium emulsion.

TREATMENT.—Removal through an incision in front of the sternomastoid. There is great danger of fatal sepsis tracking down the mediastinum. This may be minimized by: (1) Bringing diverticulum through skin opening, sewing it there, and removing later, or (2) Cutting through neck of diverticulum by diathermy cautery, invaginating into œsophagus, and sewing up flush with wall of latter

AFFECTIONS OF THE ŒSOPHAGUS

Malformations.—

CONGENITAL —

Fistulous communication with trachea.

Stricture at the cardiac orifice of the stomach.

ACQUIRED.—

TRACTION DIVERTICULA —Small pouches on anterior wall. Near tracheal bifurcation Caused by traction of adherent glands. Produce no symptoms. May cause lodgement of foreign body.

Foreign Bodies in the Œsophagus.—Coins, tooth-plates, pins swallowed accidentally, usually by children or lunatics

CHARACTERS OF FOREIGN BODY which are important·—

SIZE, causing blocking at the entrance, with fatal dyspnœa from pressure on larynx.

IRREGULARITY, causing it to catch and be impacted in the wall.

SHARP POINTS, causing perforation.

RESULTS —

PASSAGE per vias naturales, especially with small, round, smooth bodies.

IMPACTION, especially in the case of large or irregular bodies.

Position of impaction Opposite the larynx (6 inches from teeth); opposite tracheal bifurcation (12 inches from teeth); at lower end (18 inches from teeth).

ULCERATION of the gullet, resulting in cellulitis of the neck, or mediastinitis; opening into the trachea; opening into a large vessel, with fatal secondary hæmorrhage.

MIGRATION to a distance along fascial planes, in the case of pins or needles.

Foreign Bodies in the Œsophagus, *continued.*

SYMPTOMS of foreign body in œsophagus.—Dysphagia—Dyspnœa, especially if the neighbourhood of the larynx is involved.

LATE SYMPTOMS.—Pain from ulceration and inflammation—Hæmorrhage.

TREATMENT.—

LOCATION BY: Inspection of fauces—Inspection by œsophagoscope—Digital exploration—X rays.

REMOVAL via œsophagoscope. Failing that, by left-sided œsophagotomy. Attempts to remove foreign bodies with the aid of a coin catcher or probang are dangerous.

Œsophageal Obstruction.—

CAUSES.—

EXTRINSIC: Tumour in neck (e g., goitre)—Tumour in thorax (e g., aneurysm)—pressing on the gullet.

INTRINSIC: Spasm—Impacted foreign body—Fibrous stricture—Malignant stricture.

Spasm of the Œsophagus.—

HYSTERICAL spasm is only temporary.

CARDIOSPASM or achalasia is a chronic and serious disease. The lower end of the gullet as it passes through the diaphragm is affected with spasmodic contracture. The rest of the œsophagus is dilated and elongated These three points give characteristic X-ray signs after a barium meal. A full-size bougie passes easily Women usually affected with hysterical spasm, men more often with achalasia. Usully 50 years of age or over. Suggested that there is destruction of Auerbach's plexus in the œsophagus

Symptoms.—Gradual insidious dysphagia, more marked after fluids.

Treatment —By the passage of tube filled with mercury. Dilatation of œsophageal opening, by the fingers inserted through the stomach Mikulicz's operation. Transdiaphragmatic œsophago-gastrostomy has been tried with success

Fibrous Stricture of the Œsophagus.—Usually in middle-aged men. At upper or lower end of the gullet. Caused by the contraction of corrosive ulcers; possibly by syphilitic ulcers.

SYMPTOMS.—Dysphagia—Regurgitation of little altered food—Little or no bleeding on passing a sound—Slow in its development.

TREATMENT.—

PASSAGE OF TWO OR THREE SIZES OF BOUGIE once a week.

SYMONDS' TUBE —Passed on a whalebone guide. Retained, with a thread fixed to the cheek for the purpose of periodical removal.

NEW ŒSOPHAGUS.—(1) Œsophagus is brought out on to the neck; (2) Gastrostomy opening on abdomen; (3) Œsophagostomy and gastrostomy openings are joined by a rubber tube, skin tube, or piece of intestine.

DIVISION OF THE STRICTURE, followed by bougies.

From the mouth: By a concealed knife cutting backwards. Only suitable for high strictures.

From the stomach: By forcible dilatation of stricture. Only possible for stricture of lower end.

By string saw: Patient swallows a string. Lower end is pulled out of stomach. Pulled up and down.

Œsophagostomy.—When the stricture is high up. Œsophagus is sewn into left side of neck.

Gastrostomy for worst cases.

Malignant Disease of the Œsophagus.—Patients over 45; usually men.

SITUATION AND VARIETY.—

1. Opposite the Cricoid Cartilage—6–8 inches from the teeth, 15 per cent.
2. Opposite Tracheal Bifurcation.—12 in. from the teeth, 32 per cent. In both of these the growth is an epithelioma. The epithelioma presents no cell nests.
3. At the Cardiac Extremity.—18 in. from the teeth, 52 per cent. Growth is a columnar-celled carcinoma

SYMPTOMS.—Dysphagia. Feeling of weight behind the sternum. Inability to swallow solids and, later, liquids. Regurgitation of more or less altered food. This is not so marked as in fibrous stricture, because there is not time enough for dilatation above the growth. Pain and cough. Vomiting of blood. Very rapid emaciation.

PHYSICAL SIGNS.—Growth may be seen by an œsophagoscope. Auscultation detects a gurgle over the spine opposite the stricture on swallowing fluid.

EXTENSION AND COMPLICATIONS.—Early ulceration—Invasion of trachea, bronchi, great vessels—Metastatic growths in the glands are rare.

OTHER COMPLICATIONS.—Septic pneumonia, from perforation of the air-passages. Aphonia from involvement of the larynx. Laryngeal paralysis, from involvement of the recurrent laryngeal nerves. Cellulitis, or mediastinitis.

COURSE.—Death from inanition, 6–12 months.

TREATMENT.—

1. Radical Treatment —Is rather a forlorn hope, because of age and feebleness of patient, inaccessibility of growth, and difficulty of access. The most hopeful method is: (1) Division of gullet from stomach with gastrostomy, (2) Blunt separation of the gullet from its surroundings in the lower half of posterior mediastinum; (3) Exposure of gullet in the neck, separation of upper half of mediastinal connexion, withdrawal of gullet on to surface of neck, sewing to skin of neck, removal of the rest of œsophagus; (4) Making new gullet, joining the œsophagostomy (rubber, skin, or intestine).
2. Palliative Treatment —Aims at overcoming the obstruction. Methods:—
   - *a*. Insertion of a Souttar's tube (spiral of German silver wire); this is preferable to a Symonds' tube.
   - *b*. Diathermy and radium results have been disappointing, but deep X-ray therapy is more satisfactory.
   - *c*. Gastrostomy for late cases.

CHAPTER XXXI

AFFECTIONS OF THE NECK

CYSTS OF THE NECK

Congenital.—Often do not appear until adolescence.

1. DERMOIDS —

UNDER THE STERNOMASTOID —

Branchial Tubulodermoids (*Fig* 146) —May open along anterior border of sternomastoid as branchial fistulæ May become malignant—branchial carcinoma

IN MID-LINE —

Ordinary Sequestration Dermoids

Thyroglossal Duct Cysts —From thyroid duct—Foramen cæcum in tongue—Base of tongue or floor of the mouth—Behind hyoid bone —In front of larynx or trachea.

Adenomata may arise from their walls in any situation

They may open as a median fistula Generally in mid-line low down near sternum

Usually burst above or below the hyoid bone and cause a chronic sinus. The track can be demonstrated by X rays after filling with lipiodol

Treatment: By splitting hyoid bone through median vertical incision and dissecting out Liable to recur from one or more branches having been left

2. CYSTIC HYGROMA —Large shapeless mass of dilated lymph-spaces. Whole of the side of the neck and face, and down into the thorax. Not limited by any anatomical boundaries

Acquired.—

SEBACEOUS CYSTS.—Fixed to the deep skin surface.

BURSAL CYSTS —Over thyroid cartilage—Between hyoid and thyroid.

HYDROCELE OF THE NECK —Probably a lymph cyst. Under the sternomastoid, and extends into posterior triangle

BLOOD CYSTS.—Aneurysm—Venous varix—Cyst communicating with vein

GLANDULAR CYSTS —Thyroid move with deglutition Salivary: rare except in the floor of mouth.

MALIGNANT CYSTS.—From branchial dermoids—From breaking down glands the seat of secondary epithelioma—From endothelioma of carotid gland.

HYDATID CYSTS.

CUT THROAT

SUICIDAL.—Generally from left to right—More severe on left side.

HOMICIDAL.—Wounds vary in position and extent.

Varieties.—

1 Those not opening pharynx or air-passages.

2 Those involving the air-passages—Above hyoid—Through thyrohyoid space—Into larynx—Into trachea

Primary Effects.—

ABOVE HYOID —Wound of lingual and facial vessels Injury of root of tongue Danger of asphyxia from tongue falling over larynx

THYROHYOID SPACE —Wounds of lingual, facial, and superior thyroid vessels over larynx Injury of epiglottis Bleeding into larynx

LEVEL OF LARYNX —Injury to the vocal cords or lobes of thyroid gland

BELOW LARYNX —Hæmorrhage from thyroid gland and vessels—Trachea opened—Asphyxia from displacement of the cut ends or from bleeding into the trachea—Wounds of great vessels, with fatal hæmorrhage—Air drawn into veins—Division of recurrent laryngeal nerve

Remote Effects.—Septicæmia or pyæmia—Cellulitis leading to mediastinitis—Œdema of glottis—Secondary hæmorrhage—Tracheitis, bronchitis, septic pneumonia—Passage of food into trachea—Surgical emphysema

Treatment.—Arrest the hæmorrhage Sew up, with free drainage If larynx is opened, close it and perform tracheotomy If trachea is opened, insert tube If wound is above larynx sew it up, drain, and perform tracheotomy

Sequelæ to Cut Throat.—Aerial fistula, generally in the thyrohyoid space Pharyngeal fistula Laryngeal or tracheal stenosis Aphonia from injury of larynx or recurrent nerve

DISEASES OF THE THYROID GLAND

ATROPHY AND HYPERTROPHY

Atrophy is found in three conditions —

SENILE ATROPHY —Causes no symptoms

MYXŒDEMA —The epithelial tissues disappear partly or entirely, and what remains is only connective tissue

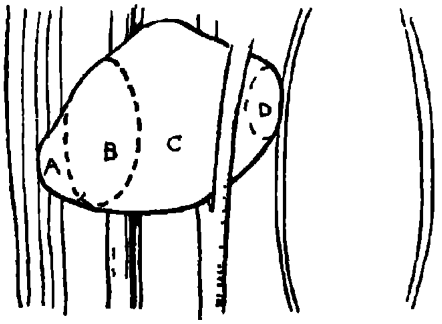


Fig 146.—Cysts in the neck derived from remains of a branchial cleft The whole cyst (C) represents the maximum extent, reaching from the sternomastoid muscle to the pharynx. A, Remnant adjoining muscle, B, Cyst between muscle and vessels, D, Cyst adjoining the pharynx. (*After Hamilton Bailey.*)

Atrophy of the Thyroid Gland, *continued.*

CRETINISM.—The gland is either: (1) Congenitally absent; (2) Represented by a mere connective-tissue rudiment; or (3) Present as a goitre, with few or no epithelial elements. It may be sporadic or endemic, the latter being common in districts where endemic goitres occur.

In both myxœdema and cretinism there occur:—

1. CHANGES IN THE SUBCUTANEOUS TISSUES.—Great increase of fat and mucoid tissue. The hair is brittle and falls out. Supraclavicular fatty masses are very conspicuous.
2. CHANGES IN THE NERVOUS SYSTEM.—Amentia or dementia is prominent. In the case of cretinism a state of childishness or imbecility continues throughout life unless the case is treated.
3. CHANGES IN THE SKELETON.—The terminal phalanges become broad and spatulous. In cretinism growth is arrested, so that the stature of the adult is that of a child of about six.
4. CHANGES IN THE GENITAL ORGANS.—The cretin has undeveloped sexual glands, undescended testes, and no secondary sexual characters. In myxœdema sexual feeling and power are lost.

TREATMENT.—

FEEDING with dried extract of sheep's thyroid (1–1½ g. twice weekly, increasing the dosage as required).

GRAFTING with human thyroid gland. Small pieces are taken from healthy thyroid tissue (e g. from a patient with a thyroid adenoma) and implanted: (1) Under the skin; (2) Beneath the abdominal muscles.

Hypertrophy (apart from parenchymatous goitre) is found in the following:—

1. Of a portion of gland when the rest has been removed.
2. During menstruation (probably merely a hyperplasia), at puberty, during pregnancy, after sexual excitement.

ENLARGEMENTS OF THE THYROID GLAND

THYROIDITIS

Varieties.—

1. ACUTE INFECTIVE —Is rare, and commoner in enlarged thyroids. May accompany the exanthemata. Gland becomes swollen, tender, and fixed.

   TREATMENT —Fomentations Incision.
2. CHRONIC.—Hashimoto type. Riedel type. Tuberculous (very rare). Syphilitic (very rare).

   HASHIMOTO'S THYROIDITIS.—Usually women 40 years and over. Obesity, early myxœdema. Gland hard, irregular, lobulated Section shows marked lymphomatous infiltration.

   Treatment.—Excision.

   LIGNEOUS (*Riedel's Disease of the Thyroid*).—A lymphomatosis, followed by a fibromatosis of the gland. Involves both lobes and isthmus. Perithyroiditis occurs, with fixation of the gland. Pressure symptoms develop.

   Treatment.—By X-ray therapy, or excision of the isthmus or one lobe.

GOITRE

SIMPLE OR PARENCHYMATOUS ENLARGEMENT

In this the enlargement is uniform. It is the common form in young patients, and rarely begins after twenty-five. (*Fig.* 147.)

Stages in Development.—(1) Hypertrophy of the vesicles: goitre is then hard and solid. (2) Colloid stage: excess of colloid distends the vesicles. (3) Colloid cystic stage: vesicles run together and form cysts. (4) Stage of focal hyperplasia or nodular goitre: little islands of fœtal adenomatous cells in the interacinous spaces multiply to maintain the function of the gland that is otherwise being destroyed. (5) Degenerative goitre: areas of degeneration, areas of calcification, areas showing hæmorrhage into cysts.

Aetiology of Parenchymatous Goitre.—It is usually endemic. The localities most affected are in Switzerland, mountainous districts of France and Italy, Derbyshire and Gloucestershire in England. It is associated with a limestone geological formation, which probably causes some peculiarity of the drinking-water. Is not due so much to iodine deficiency, but to a combination of factors by which the tissues are unable to utilize iodine even when present in normal amounts.

Physical Signs of Goitre.—

MOBILITY.—The gland moves up and down in swallowing. It cannot be separated laterally from the trachea. This alone usually suffices for the diagnosis, but:—

1 OTHER CERVICAL SWELLINGS WHICH MOVE ON DEGLUTITION are: Lymph-glands adherent to the trachea; abscesses or malignant growths connected with larynx, trachea, or œsophagus; subhyoid bursæ; thyroglossal cysts.

2 GOITRES WHICH DO NOT MOVE WITH DEGLUTITION: Very large masses which cannot pass through the thoracic inlet. Goitres which are fixed by malignant or inflammatory adhesions.

SHAPE AND SIZE.—Parenchymatous goitres may be horse-shoe-shaped, but others are usually oval. The size is anything up to that of a man's head, it hangs over the sternum.

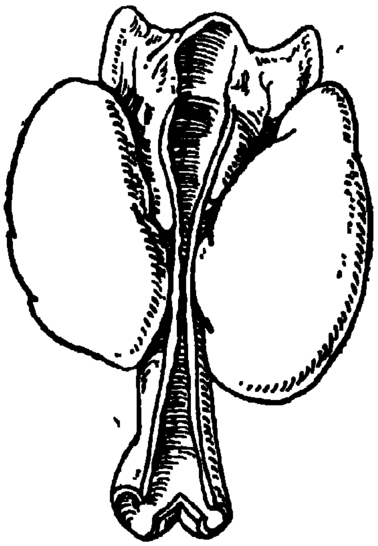


Fig. 147.—Parenchymatous goitre seen from behind. The trachea is shown cut open, and is compressed laterally between the enlarged thyroidl obes.

Simple Goitre—*Physical Signs, continued.*

POSITION.—Usually below the larynx. In the mid-line or lateral. Sometimes substernal; rarely high up on a level with the great hyoid cornu. Pushes the sternomastoid muscle outwards.

RELATION TO VESSELS.—The common carotid is pushed backwards and outwards, whilst the internal jugular vein becomes spread over its surface, being tied to it by the thyroid veins. In malignant goitre the vessels become surrounded without much displacement.

RELATION TO THE STERNUM —Occasionally substernal goitres descend as low as the arch of the aorta.

PULSATION.—Thyroid tumours often present a pulsation, which is either (1) communicated from the carotid, or (2) is that of the thyroid arteries, or (3) is due to the vascular nature of the growth, especially in Graves' disease. A bruit is often heard—loud and rasping—in these cases.

CONSISTENCE.—Soft when parenchymatous. Firm and elastic when adenomatous or cystic Hard when fibrous or calcified.

PRESSURE EFFECTS.—

ON VEINS.—The jugular veins become engorged, but œdema and cyanosis are seen only in malignant or inflamed growths.

ON NERVES.—The recurrent laryngeal, sympathetic, cervical, and brachial nerves may be involved, in this order of frequency, but marked nerve involvement is a grave sign of malignancy .

ON THE TRACHEA —This may be (1) displaced laterally, (2) kinked, or (3) compressed laterally, so that its cavity is reduced to a chink. This is the common cause of dyspnœa

ON THE ŒSOPHAGUS.—Very rarely a posterior thyroid growth may cause displacement and compression of the gullet

Dyspnœa.—This is practically the only symptom produced by goitres. It arises in several ways, pressure on the trachea being far the commonest:—

1. TRACHEAL PRESSURE —The trachea is displaced laterally, or sometimes backwards (by a substernal goitre) Its lumen is narrowed by (*a*) kinking, (*b*) unilateral pressure, (*c*) bilateral pressure (*see Fig.* 147). The typical scabbard trachea can be demonstrated by an antero-posterior radiograph It becomes soft and yielding after long pressure, but not actually atrophied.
2. BY PRESSURE ON THE RECURRENT LARYNGEAL NERVES.—This is very rare, as shown by the infrequence of aphonia combined with dyspnœa.
3. BY EXTENSION OF THE GROWTH INTO THE AIR-PASSAGES. —This occurs only in malignant disease
4. BY RUPTURE OF A CYST OR ABCESS INTO THE TRACHEA.
5. BY CAUSING ŒDEMA GLOTTIDIS.—Only in inflamed or malignant goitres.
6. BY SWELLING OF THE MUCOUS MEMBRANE of the air-passages. This occurs when the patient catches cold or contracts bronchitis.

INCIDENCE OF DYSPNŒA IN GOITRE.—It occurs most often in patients between the age of puberty and twenty, in parenchymatous, bilateral, and substernal goitres.

ITS SUDDEN APPEARANCE may be due to hæmorrhage into a cyst, or the rapid enlargement of cystic cavities.

A BRASSY COUGH generally accompanies the dyspnœa, and is due to the tracheal affection.

Treatment of Simple Goitre.—

GENERAL.—Removal of the patient from goitrous district and the addition of hygienic measures, elimination of septic foci, and the administration of syr. ferri iodidi ʒi t d.s. No iodine or thyroid extract necessary. This drug treatment only acts in parenchymatous cases in young patients. It has no effect on cysts or adenomata. Marked diminution will occur in one month or not at all.

INDICATIONS FOR OPERATION.—Dyspnœa or other pressure signs, or steady enlargement Cysts or adenomata. Failure of medicinal treatment in diffuse goitre.

PRECAUTIONS.—Use of local anæsthetic in those cases where dyspnœa is well marked. Intratracheal anæsthesia overcomes all difficulties due to dyspnœa, and by minimizing respiratory movements it gives a very placid operation. Performance of the operation in the position of greatest ease as regards breathing.

OPERATION.—

1 **Partial Thyroidectomy**—One-half or more of gland is removed in its capsule through a collar incision. Infrahyoid muscles are drawn outwards or cut. Leaves a large unsightly unilateral lobe.

Special care is to be taken of (1) the internal jugular vein, as it lies expanded over the goitre, and (2) the recurrent laryngeal nerve, as it lies behind and in very close connexion with the tumour below the cricoid cartilage.

Divide the isthmus and remove the lobe Drain for twenty-four hours.

2. **Bilateral Wedge Resection.**—The operation of choice in diffuse goitre. A large wedge with base anteriorly is cut from each lateral lobe, and the cavity left is obliterated by catgut suture. The isthmus is divided between clamps

3 **Resection and Enucleation** is the operation for cysts and adenomata, which are shelled out from a capsule formed by thinned gland substance

ACCIDENTS WHICH MAY OCCUR DURING THYROID OPERATIONS.—

Sudden Death from dyspnœa occurs in cases where severe dyspnœa is present at the outset Due to traction on, or pressure on, the trachea, the general anæsthetic, spasm of the glottis, or traction on the nerves in the neck. It is most liable to occur when the tumour is dragged on or turned out of its bed It is best avoided by local or intratracheal anæsthesia.

Primary Hæmorrhage.—This is more likely to be severe in enucleation operations. In extirpation all the chief vessels should be tied before they are cut. It is chiefly venous bleeding that gives trouble.

Injury of the Recurrent Laryngeal Nerve.—Causes unilateral vocal paralysis

Injury of the Sympathetic or Vagus.—The latter may cause death.

Injury of the Trachea, Pleura, Œsophagus, or Pharynx.

Collapse of the Flattened Trachea.—This is very rare, but may require tracheotomy.

ACCIDENTS WHICH MAY OCCUR AFTER THYROID OPERATIONS.—

Recurrent Hæmorrhage—Especially after enucleations and if adrenaline solution has been used with a local anæsthetic. It may

Simple Goitre—Treatment, *continued.*

cause death from hæmorrhage or from dyspnœa. It is a wise precaution to let patient come partly round before sewing up.

SEPSIS, leading to cellulitis or mediastinitis, or to a chronic sinus.

RESTLESSNESS AND RAPIDITY OF PULSE AND RESPIRATION.—This is most often seen in operations for toxic goitre, next in parenchymatous goitres It is probably due to rapid absorption of thyroid secretion. It may be fatal. It is best avoided by careful handling of the tumour, free irrigation of the wound with water, and drainage.

LATE VOCAL PARALYSIS, due to involvement of the recurrent nerve in scar tissue.

TETANY.—Very rare Probably due to removal of the parathyroid glands

REMOTE COMPLICATIONS.—Only seen after total thyroid removal.

CACHEXIA STRUMIPRIVA —Symptoms develop two or three months after operation Fatigue is complained of, and the limbs are heavy and indolent. The hands become swollen and clumsy, and all fine movements impossible. Skin is pale and much swollen, especially over the eyelids, forehead, cheeks, and tongue, but there is no pitting on pressure The facial expression is dull and vacant. Cerebration is very slow, and memory is bad Speech is slow and laboured. The skin becomes dry and scaly, and the hair falls out The temperature is subnormal, and there is great sensitiveness to cold If the patient is immature, growth and sexual development are arrested. In adults sexual functions cease

Treatment —Administration of thyroid extract, or thyroid grafting.

TOXIC GOITRE

There are two varieties of this condition (1) Primary thyrotoxicosis (Graves' disease), (2) Toxic adenoma Respective characteristics:—

Primary Thyrotoxicosis	*Secondary Toxic Goitre or Toxic Adenoma*
1. In early life.	1 In later life.
2. Symptoms commence within one year of enlargement of gland.	2. May be no symptoms for 10 or 12 years after enlargement.
3. After two or three months may improve and later relapse.	3. Symptoms, once they appear, are always progressive.
4. Exophthalmos almost always present.	4. No exophthalmos.
5. May be arrested in development by medical treatment. May or may not cause death from tachycardia and failure in the heart muscle action. May pass to hypothyroidism.	5. If not removed by operation, death always results from tachycardia, and cardiac muscle failure. Does not yield to medical treatment.
	For remaining symptoms see section on PRIMARY THYROTOXICOSIS (*below*).

Primary Thyrotoxicosis.—

AETIOLOGY.—Females form 90 per cent of the cases, and the age incidence is the early period of sexual activity (25–40, usually before 35 years), and frequently following a crisis in the patient's life. The local occurrence bears no relation to endemic goitre.

ANATOMY.—The gland is uniformly but not very greatly enlarged. It is of smooth surface and firm fleshy texture. The blood-vessels are not large, but are greatly increased in number. Microscopically the tissue shows great epithelial proliferation and diminution or absence of colloid material. The thymus gland is often enlarged.

PATHOLOGY.—The disease is due to: (1) Increased thyroid activity; (2) Perverted thyroid secretion; (3) Increased absorption of thyroid secretion. Evidence of causative disease of the central nervous system or sympathetic is quite absent.

SYMPTOMS.—There are acute exacerbations and remissions.

CARDINAL —(1) Uniform and slight thyroid enlargement. (2) Prominence of the eyeballs, with delay in the descent of the upper lid when shutting the eyes, widening of palpebral fissure so that there is a white margin all round the corneal rim (Stellwag); delay in dropping of the upper eyelid when looking down (Von Graefe), inability to converge when looking at a close object (Moebius); absence of forehead wrinkling as patient looks up (Joffroy). (3) Tachycardia, with attacks of palpitation and auricular fibrillation.

ACCESSORY.—(4) Nervous symptoms, tremor, excitability, and attacks of acute mania; (5) Sweating, with intolerance of heat; (6) Dyspnœa; (7) Loss of weight, (8) Diarrhœa, (9) Glycosuria; (10) Patchy pigmentation; (11) Local fatty swellings, especially on the abdominal wall; (12) Slight elevation of temperature.

INCREASE OF BASAL METABOLISM.—The amount of oxygen used in a given time under resting conditions is measured, and compared with that of a normal individual of the same age, weight, height, and sex. Any variation of over 10 per cent from the normal shows altered metabolism. In exophthalmic goitre the rate is increased from 20 to 60 per cent The amount of this increase is a good index of the gravity of the disease.

OCCASIONALLY the goitre or the exophthalmos may be absent. The exophthalmos is due to an accumulation of fat in the orbits and to spasm of the unstriped muscle fibres which lie at the back of the eyeball. The dyspnœa is due to cardiovascular changes, and is not accompanied by stridor

TREATMENT —

MEDICAL —Depends on the severity of the symptoms, clinical picture, and the basal metabolic rate.

Strict régime Rest in bed, light nourishing diet, fluids, and glucose. Luminal, g. ½ b. d., and bromides are of great value in controlling the nervous symptoms

Cardiovascular conditions are treated by routine methods.

Lugol's Iodine —Of great value when administered at correct time. Produces a 'constipation' of the thyroid, causing it to store up its perverted secretion and so prevent it having effects on the various organs. Given in doses of ♏x t d s, increasing to ♏xv–♏xx depending on clinical condition. Maximum effect in 10–12 days, at which period operative procedures should be performed.

RADIOTHERAPY.—X rays or radium may produce reduction in the size of the goitre, but are rarely curative.

SURGICAL —

Arterial Ligature —Both superior and one inferior arteries are tied. Of value in severe cases.

Primary Thyrotoxicosis—Treatment, ***continued.***

Subtotal Thyroidectomy.—Removal of approximately three-quarters to seven-eighths of the gland. The posterior portion of each lobe is left, so as to save the parathyroids and to avoid the recurrent laryngeal nerves Gentle handling and meticulous care in hæmostasis are essential. Drainage of wound for 48 hours. Prior to operation great care must be taken with treatment. Complete rest and quiet. The heart to be fortified by previous treatment, which must be continued after operation. Basal metabolism should approach the normal if possible. The patient should be in a resting phase of the disease The alarm of the operation should be minimized This may be done in various ways. Rectal ether with a previous preparation by simple enemata Twilight sleep, with removal under local anæsthetic, or intratracheal ether.

Post-operative Treatment—Prop up in bed; sedatives Glucose in saline rectally or intravenously. Lugol's iodine ʒi in milk per rectum following operation. Lugol's iodine ♏x t d s for 5 days after operation Patients kept cool by a fan

Toxic Adenoma.—A localized adenomatous growth in one lobe of the thyroid. Usually single and of slow enlargement. May arise in a normal gland or in pre-existing goitres and adenomata Its clinical characteristics are given in the above table.

TREATMENT —Removal by operation essential, with precautions similar to those taken in primary thyrotoxicosis.

NEW GROWTHS

ADENOMA

Features.—

Usually solitary, though two or three may occur, usually at junction of isthmus and lateral lobe or retrosternal

Age 25–45

Two types (1) Fœtal—solid masses of tubular acini, no colloid, no lumen; (2) Cystic

If untreated may become (1) Toxic, (2) Calcified, (3) Malignant; (4) Infected, (5) Develop a sudden intracystic hæmorrhage.

Toxic adenoma gives tachycardia and nervous symptoms, but never exophthalmos

Treatment.—Resection and enucleation.

MALIGNANT DISEASE

Aetiology.—Rarely occurs before forty. Affects both sexes equally. Often develops in a simple goitre

Structure.—Alveolar carcinoma and sarcoma, either round- or spindle-celled, are of about equal frequency Sarcoma is rather more likely to form localized swellings of rapid growth, and carcinoma general enlargement of slower growth, but usually the distinction is impossible.

Symptoms and Signs.—Special signs of a goitre being malignant are: (1) Hardness, with bossy outline; (2) Evidence of early nerve involvement —unilateral vocal cord paralysis, shooting pains up the neck and down the arm, contraction of the pupil and palpebral fissure; (3) Involvement of blood-vessels—the carotid, instead of being displaced, is surrounded by the growth, and cyanosis with œdema of the face may occur late in the case from obliteration of the veins; (4) Penetration of the trachea, and much more rarely the pharynx, by the growth; (5) Great fixity to the surrounding structures The skin and lymph-glands are, however, rarely involved.

Types.—Types of carcinoma found are: (1) Malignant adenoma 80 per cent; (2) Papilliferous adenocarcinoma 16 per cent; (3) Scirrhous carcinoma 4 per cent.

Metastasis occurs in the bones and the lungs. In the former case pulsating tumours are formed, especially on the cranial bones. Metastasis sometimes occurs from thyroid tumours which show no other sign of malignancy. Metastases still function and produce internal secretion.

Death occurs in three months to two years.

CAUSES.—(1) Dyspnœa; (2) Penetration of the trachea with septic pneumonia; (3) Œdema glottidis, (4) Hæmorrhage into the growth, with sudden dyspnœa

Adenomata and Papilliferous Adenomata.—Adenomata when they become malignant, have only a limited malignancy. that is, they are very liable to recur, but at first do not infiltrate the tissues or cause metastases

Treatment.—Radium or deep X rays.

RADICAL.—Is only possible in a small proportion of cases; often involves resection of the great vessels or trachea Operative mortality is 35 per cent, and survival for three years only 6 per cent

PALLIATIVE.—Local removal of masses which compress the trachea

TRACHEOTOMY.—This is rarely possible below the tumour Often it has to be done through the growth Generally it should be done above the growth, and a long Koenig's tube, 4 to 5 in. long, passed down beyond it

THE PARATHYROIDS

These are four small bodies about ¼ in in diameter, arranged in pairs behind the thyroid gland They are very variable in size, position, and number. If they are removed in thyroid operations, TETANY, which is usually fatal, results This condition is to be treated by calcium and parathyroid extract.

(*See also* GENERALIZED OSTEITIS FIBROSA, p 227)

CHAPTER XXXII

DISEASES OF THE AIR-PASSAGES AND CHEST

FOREIGN BODIES IN THE AIR-PASSAGES

In Nasal Passages.—Common in children. Unilateral purulent discharge.

TREATMENT.—Removal under an anæsthetic is often necessary on account of swelling present.

At the Rima Glottidis.—Food mass, or large foreign body. Rapid asphyxia and death

TREATMENT.—Removal by the finger from the mouth, or immediate laryngotomy.

In the Larynx.—Small, round, foreign bodies. Produce urgent dyspnœa; croupy cough Complete obstruction occurs later from œdema.

TREATMENT —Remove by laryngeal forceps, laryngotomy, or thyrotomy. Foreign body must be gripped firmly otherwise there is a tendency for it to fall down into the trachea or bronchus.

In the Trachea.—Attacks of spasmodic cough and dyspnœa caused by the foreign body impacting against the larynx. Tracheitis, with cough and expectoration

TREATMENT.—Removal through a bronchoscope. Low tracheotomy, with removal.

In a Bronchus.—Smooth, round, heavy bodies, e.g., marble or intubation tube. Generally into the right bronchus, because: It is more in a line with the trachea; the septum between the bronchi lies to the left of the middle line; the right bronchus is larger than the left in the proportion of five to four.

SYMPTOMS —Short attack of spasmodic dyspnœa, due to the passage through the larynx. Collapse of the corresponding lung, due to the air escaping more easily than it enters. Produces: (1) Dullness and absence of breath-sounds; (2) Unilateral bronchitis, bronchiectasis; (3) Abscess of the lung, or pneumonia, (4) Empyema.

RESULTS —Death from one of the above. Expulsion with pus from an abscess by violent coughing. Expulsion with the pus of an empyema.

TREATMENT.—Removal through a bronchoscope; removal through low tracheotomy or by thoracotomy.

LARYNGEAL AFFECTIONS

Œdema Glottidis, or Œdematous Laryngitis.—

CAUSES.—

1. DIRECT INJURY of larynx by scalds, corrosives, or wounds.
2. FOREIGN BODY impacted in the larynx.
3. SECONDARY to: (*a*) Any inflammatory disease of the larynx; (*b*) Cellulitis of the neck (Ludwig's angina); (*d*) Acute glossitis; (*e*) Retropharyngeal abscess.

ANATOMY.—Swelling and œdema of: aryteno-epiglottidean folds; inter-arytenoid fold; epiglottis, which becomes folded on itself; false vocal cords. Rima glottidis becomes partially or totally occluded.

SYMPTOMS AND SIGNS —Intense inspiratory dyspnœa. Epiglottis and folds above the larynx are seen and felt to be swollen, red, and œdematous.

TREATMENT.—

IN SLIGHT CASES.—Scarification of the swollen mucous membrane. Ice compresses externally.

IN BAD CASES.—Tracheotomy.

Diphtheritic or Membranous Laryngitis.—

SYMPTOMS.—Usually associated with faucial diphtheria, which is recognized by the yellow membrane. Rapidly causes dyspnœa, evidenced by crowing inspiration, lividity, and recession of the lower part of the chest.

TREATMENT.—Intubation or tracheotomy as soon as chest recession appears.

TRACHEOTOMY is the usual operation. Lightest possible anæsthesia. Head to be held quite straight, neck hyper-extended over a sand-bag. Identify the cricoid cartilage Median vertical incision over and below cricoid. Hold cricoid cartilage with sharp hook. Expose front of trachea and cut upper two rings of trachea. Insert dilators and then tracheotomy tube, which is tied in by tape.

INTUBATION, i e., the insertion of a special metal tube into larynx, is only suitable in large institutions when the number of cases affords sufficient experience.

Syphilis of the Larynx.—

IN THE SECONDARY STAGE —Mucous tubercles Superficial ulceration. Producing hoarseness and aphonia

IN THE TERTIARY STAGE —Gummatous infiltration, and deep destructive ulceration affecting: (1) The epiglottis, which may be completely destroyed, (2) The parts above the true vocal cords. Necrosis of cartilages. Perichondritis Later, laryngeal stenosis.

SYMPTOMS.—Early: hoarseness of the voice, hoarse cough, aphonia. Late dyspnœa from stenosis

TREATMENT.—Iodides and mercury Tracheotomy for stenosis. Iodides to be used with caution if stenosis is present

Tuberculous Laryngitis.—Primary, or secondary to phthisis. Lupus.

ANATOMY —Tuberculous infiltration and ulceration of arytenoid elevations, aryteno-epiglottidean folds, and epiglottis.

SYMPTOMS.—Great pain—Hoarseness and cough—Dyspnœa and painful dysphagia.

TREATMENT.—

GENERAL.—Open air in warm climate, and forced feeding. Absolute silence.

LOCAL.—Inhalations of orthoform powder for pain. Curetting the tuberculous deposits after cocainization, and then rubbing in lactic acid (50 per cent solution).

Laryngeal Affections, ***continued.***

New Growths of the Larynx.—

PAPILLOMA.—In young subjects—Generally arises from the vocal cords—Causes hoarseness, dyspnœa, or laryngeal spasm.

TREATMENT.—Removal by intralaryngeal cutting forceps.

FIBROMA and ADENOMA also occur, but are very rare.

MALIGNANT TUMOURS:—

Classified as: (1) Intrinsic tumours—vocal cords, ventricular bands, etc. (2) Subglottic tumours; (3) Extrinsic tumours—epiglottis, pyriform sinus; (4) Mixed tumours.

Malignant tumours usually over 40 years of age if squamous-cell carcinoma, though basal-cell tumours may occur

1. INTRINSIC TUMOURS —Anterior portion of vocal cord, spreading slowly along it. Lymphatic glands invaded late.

   Symptoms.—Hoarseness, cough, pain, and dyspnœa later when growth has spread across to other side Later growth becomes extrinsic with perichondritis and dysphagia.

   Differential Diagnosis.—Simple tumours, chronic laryngitis, syphilis. Fixation of cord, though an important sign, is a sign of infiltration of the cord

   Treatment —Excision by laryngofissure. Late cases—radium.

2. SUBGLOTTIC TUMOURS.—Insidious with signs of laryngeal œdema. May spread through crico-thyroid membrane to lymph glands. Produce paresis of the cord

   Treatment.—Laryngectomy

3. EXTRINSIC TUMOURS —Epiglottis and aryteno-epiglottidean folds Local discomfort; later pain in jaw and ear Local glandular spread and progress is rapid In females a post-cricoid carcinoma is common, producing symptoms of dysphagia

   Treatment.—Excision by lateral pharyngotomy Dissection of glands of neck Deep X-ray therapy, temporary improvement only Teleradium.

4. MIXED TUMOURS —Usually inoperable Tracheotomy is of value to relieve dyspnœa.

SURGERY OF THE CHEST

Injuries and Wounds of the Thorax.—These are usually divided into (1) Non-penetrating; and (2) Penetrating.

1. NON-PENETRATING WOUNDS —Severity depends on: (*a*) Nature of force causing injury; (*b*) Type of force—either sudden or gradual; (*c*) Condition of lung at time of accident, (4) Age of patient an resilience of thoracic structures

   Severe injuries result from compression of chest either between two objects or in 'run over' accidents. May be complicated by rupture of liver, spleen, or kidneys, or by traumatic laceration of diaphragm; also associated with fractures of ribs, sternum, or scapula.

   CLINICAL FEATURES:—May be three: (i) Shock; (ii) Hæmorrhage; (iii) Pneumothorax or surgical emphysema.

   Hæmorrhage may result from a laceration of the lung or subcostal or internal mammary artery. Signs of pleural effusion, associated with signs of internal hæmorrhage.

Surgical emphysema occurs if lung which is adherent to chest wall is lacerated.

TREATMENT.—Treat shock with morphia, rest, and oxygen. Needling of chest to confirm hæmothorax, careful watch on blood-pressure to see whether hæmorrhage is continuing. Thoracotomy may be needed if hæmorrhage severe. Treat associated injuries.

2. PENETRATING WOUNDS.—Following stab wounds or gunshot wounds. Shock is considerable. Hæmoptysis occurs. Large opening in the chest wall produces mediastinal flutter, dyspnœa, cardiac embarrassment; engorgement of veins of neck is often evident.

TREATMENT—Depends on the urgency of symptoms Arrest hæmorrhage, close wound with pad or few sutures over a pad; treat shock; when condition improved, wound explored and clots removed from pleural cavity; lung sutured if necessary; sulphonamides to wound; closure of edges after adequate excision.

Wounds of the Lungs.—

CAUSES —Contusions—Fractured ribs—Penetrating wounds.

RESULTS —Contusion or laceration of the lung may cause:—

SEVERE PAIN, SHOCK, AND DYSPNŒA.

HÆMOPTYSIS —Expectoration of blood-stained mucus or pure blood, with death from asphyxia or syncope.

HÆMOTHORAX —Rapidly increasing area of dullness, with diminished breath-sounds, without evidences of inflammation.

PNEUMOTHORAX, with pulmonary collapse.—Severe dyspnœa, with tympanitic chest note and amphoric breathing.

HÆMOPNEUMOTHORAX.—As above, but associated with splashing sounds

EMPHYSEMA.—(1) *Surgical*: Due to the air being forced across the pleural cavity, or sucked into an external wound, into the cellular tissue of the parietes (2) *Interstitial* Very rare The air enters the connective tissue of the lung, and may appear in the cellular tissue of the neck

PLEUROPNEUMONIA, or septic pneumonia

GANGRENE of the lung.

PLEURISY OR EMPYEMA, especially in cases of penetrating wounds.

TREATMENT OF LUNG WOUNDS.—Absolute rest and recumbency. Avoidance of talking Firm strapping and bandaging of the chest, unless the dyspnœa is so urgent as to forbid this

FOR HÆMOPTYSIS.—Ergot, adrenaline; these both cause contraction of the vessels and raised blood-pressure Opium and amyl nitrite act by diminishing the blood-pressure.

FOR HÆMOTHORAX AND PNEUMOTHORAX —Treat by aspiration.

FOR PENETRATING WOUNDS —Careful treatment of the parietal wound. Examination by X rays to determine foreign bodies or depressed fractures. Intercostal hæmorrhage must be arrested by enlarging the wound and removing part of the rib if necessary. Pulmonary hæmorrhage is usually beyond treatment if severe. In moderate cases it ceases when syncope occurs.

DIRECT SUTURE OF THE LUNG is difficult, because of the great tendency of the lung to retract. The production of a pneumothorax in some cases will in itself serve to check the bleeding, because the vessels share in the general collapse of the lung.

Wounds of the Lungs—Treatment, ***continued.***

Removal of Foreign Bodies is only to be undertaken: (1) If they are causing definite inflammatory symptoms; (2) If the X rays show that they are in accessible position.

Operation on the Lungs.—Special differential pressure apparatus is unnecessary Free opening of one pleural cavity with retraction of the lung is without danger. Incision is made along the 4th rib and cartilage in front, the cartilage is cut by a V-shaped incision, the periosteum is incised, and the rib held out of the way. The pleura is opened, the 3rd and 5th ribs are retracted from one another. The hand is inserted, the foreign body palpated, or the affected part of the lung is brought up into the wound. Removal of the projectiles and suture of the lung are performed. The lung is dropped back, the 4th rib is replaced, and the wound sutured.

Tumours of the Mediastinum.—

INNOCENT.—Commonest are Dermoids and Teratomas. Generally originate in mediastinum May ulcerate into bronchus, and sebaceous matter or hairs may be expectorated.

Tumours of thyroid occur retrosternally, causing pressure symptoms.

Thymus: May get simple enlargement or a true thymoma, the latter being malignant, with a varying rapidity of growth

Lipomata, chondromata, and fibromata occur, ganglion neuromata in the costovertebral groove, and neurofibromata. The last may be present as the 'dumb-bell tumour,' being partly in and partly out of the thoracic cavity

Tumours of the Lung and Bronchus.—

CLASSIFICATION: (1) Simple: Endothelioma of pleura (2) Malignant: (*a*) Primary—Bronchial carcinoma; Parenchymatous; squamous-cell carcinoma. (*b*) Secondary—Sarcoma, hypernephroma, and testicular neoplasms

BRONCHIAL CARCINOMA.—In men three times more than women, age 40–60 years. Usually in the larger bronchi, at division of main bronchi into secondary bronchi. Right lung more than left.

Pathology.—All primary carcinomata are thought to be bronchogenic in origin, originating in basement membrane of the bronchial epithelium.

Signs and Symptoms.—Cough, pain in chest, and slight hæmoptysis; loss of weight and failing health. Blood-stained pleural effusion may occur. Pressure on veins is a late manifestation. Pressure on nerves may occur. Clubbing of fingers occasionally seen.

Radiography.—Diagnostic in later stages after extension from hilum. Bronchoscopy: if tumour seen, operability is usually poor as it is too near the tracheal bifurcation. Aspiration biopsy through the chest wall is sometimes used. Examination of wet film of sputum gives a high proportion of positive results. Bronchography in some cases demonstrates bronchial obstruction with typical rat-tail appearance.

Treatment.—Depends on site, nature, and presence or absence of metastasis, and may be radical or palliative. Radical treatment may necessitate lobectomy or pneumonectomy. Palliative treatment—radium or deep X rays.

PARENCHYMATOUS CARCINOMA.—Usually upper lobe, and is a squamous-cell carcinoma. Symptoms slow in appearance, and may be delayed until pleural involvement occurs. Cough and sputum only occur if bronchus is eroded.

TREATMENT —Pneumonectomy, if patient's condition satisfactory.

Chronic Pleural Effusion.—

CAUSES.—Tubercle and heart disease. Other infections almost invariably lead to empyema

PHYSICAL SIGNS —Side of the chest moves badly. Massive dullness extending from the base upwards. Diminished breath-sounds and vocal fremitus. An area of ægophony towards the upper level of the fluid. Displacement of the heart to the opposite side, and of the liver and diaphragm downwards

SYMPTOMS are those of the primary lung condition—dyspnœa, cough, rapid pulse, etc.

SURGICAL TREATMENT.—Aspiration through the 6th interspace in the mid-axillary line. As much fluid as possible is withdrawn until coughing or bleeding begins.

Acute Empyema.—

CAUSES.—Injuries, wounds, pleuropneumonia, tubercle, abdominal suppuration. Extension from neighbouring abscesses, e.g., liver, kidney, subphrenic, etc.

MICRO-ORGANISMS —The pneumococcus is far the commonest, Tubercle is associated with the very chronic cases Staphylococci and streptococci are fairly frequent.

SIGNS.—The same as those of a serous effusion, but if left to itself, the intercostal spaces begin to bulge, and actual pointing of the abscess occurs usually in one of the upper spaces in front (empyema necessitatis).

SYMPTOMS.—As in the case of an effusion, but a raised or hectic temperature is the rule.

RESULTS —The lung becomes collapsed and fibrotic if not treated, and the visceral pleura then ties it down by becoming thick and contracted. The parietal pleura becomes enormously thick and fibrous. Permanent displacement of the heart and mediastinum, which become dragged over to the diseased side. The opposite lung undergoes some compensatory hypertrophy. The diaphragm and abdominal viscera are displaced upwards. The chest wall falls in somewhat, and the spine is curved with the concavity towards the disease.

PROGNOSIS.—

PNEUMOCOCCAL INFECTIONS run a favourable course without becoming chronic

PYOGENIC AND TUBERCULOUS CASES usually become chronic, and require resection operations to close the cavity.

STREPTOCOCCAL INFECTIONS have the worst prognosis.

TREATMENT —If ASPIRATION shows that the fluid is pus, then except in small effusions with pneumonia:—

DRAINAGE ought to be performed. First examine specimen of pus. If pneumococcal (adherent), proceed at once to provide drainage. If streptococcal (non-adherent), treat first by aspiration and then drain. Resect 2 in. of the 8th rib in mid-axillary or 9th rib in scapular line.

Acute Empyema—Treatment—Drainage, ***continued.***

Place patient on diseased side, and rather over on his face. Either use open drainage with a large tube, or closed drainage using a flanged tube of the Tudor Edwards type. In debilitated subjects intercostal drainage may be used, rib resection being performed later if necessary.

ENCOURAGE THE EXPANSION OF THE LUNG.—This may be done in two ways, which should be adopted as early as possible after the acute symptoms have abated: (1) The use of closed drainage with continuous negative pressure; (2) Making the patient blow into two Woulfe's bottles containing a quart of fluid. The fluid is driven alternately from one bottle to another, and the increased intrabronchial pressure expands the lung. It is specially suitable for children, who regard this as an amusement. Breathing exercises are of great value in promoting re-expansion of the lung

Chronic Empyema.—Usually the result of delay in treatment of acute cases, or faulty drainage. The causes may lie in:—

1 CHEST WALL.—(*a*) Inadequate drainage, either too small, not dependent, or loculi are present, (*b*) Fibrosis of chest wall and thickening of pleura.

2. PLEURAL CAVITY —(*a*) Retained foreign bodies such as safety-pins and tubes. Sequestra and tuberculosis.

3. LUNG.—(*a*) Fibrosis and thickened visceral pleura; (*b*) Bronchial fistula

The chronic undrained empyema may exist *ab initio*.

DIAGNOSIS AND TREATMENT.—Ascertain cause of chronicity by routine clinical examination, investigation of size and site of cavity by X rays and lipiodol. Bacteriological examination of the discharge. Treatment directed to the cause, e g, remove foreign bodies; open up sinus either by dilatation or further resection of ribs If no bronchopleural fistula present, irrigation with Dakin's solution and breathing exercises is of great value and may cure some early cases. If after reasonable time no improvement, or condition is stationary, then a partial or complete thoracoplasty is needed, with mobilization of a flap of thickened parietal pleura.

Bronchiectasis.—

ANATOMY.—The branches of the bronchial tree are dilated in a fusiform or cylindrical manner Usually is confined to one lobe (lower) on one side Some degree of atelectasis or fibrosis of the lung. Frequently, pleuritic adhesions

SYMPTOMS —Cough with profuse and foul expectoration. Sputum may be as much as one pint daily. It separates into three layers on standing. Ill nutrition and general malaise.

COMPLICATIONS.—Cerebral abscess, empyema.

SIGNS.—Loss of resonance at the base of chest. Coarse râles.

BRONCHOGRAPHY.—Lipiodol is injected into each bronchus separately. X rays show the position and extent of bronchial dilatation.

TREATMENT.—

POSTURAL DRAINAGE, i.e., keeping the body head downwards for several hours a day.

LOBECTOMY.—If only one lobe is affected. The chest is opened through one intercostal space. The affected lobe is separated from adhesions. A stout ligature is passed over the lobe and drawn tight by a special clamp. The lobe is removed. All vessels and bronchi are ligatured, before removing the snare. Pneumonectomy if more than one lobe affected in unilateral abscess.

Abscess of the Lungs.—

CAUSES.—Pneumonia—Gangrene—A foreign body—Wounds—An infective embolus.

SIGNS.—Are those of local consolidation, with hectic temperature. X rays give a shadow. The abscess may rupture into a bronchus, with the expectoration of a quantity of foul pus. After this there will be the physical signs of a cavity in the lung, with coarse moist bubbling râles and amphoric breathing.

Tomography is useful in localization of abscess

TREATMENT.—Free opening through the chest wall, with excision of a piece of rib. If pleural adhesions are firm, the lung can be opened directly after plunging a probe or sinus forceps into the cavity. Drain with a large tube.

If pleural adhesions do not exist, it is best eithes to stitch the visceral and parietal pleuræ together, or to pack in some iodized gauze and wait for two or three days and then open with diathermy.

Hydatid of the Lung.—This is treated in the same way as abscess.

Tuberculosis of the Lungs.—Surgical treatment is almost confined to unilateral cases. All methods aim at obliteration of diseased cavities and production of rest. They are.—

1. PRODUCTION OF ARTIFICIAL PNEUMOTHORAX
2. AVULSION OF PHRENIC NERVE.—Results in paralysis of one-half of the diaphragm.
3. THORACOPLASTY

The Production of an Artificial Pneumothorax.—

INDICATIONS.—Phthisis, in cases where the disease is chiefly unilateral. Some cases of bronchiectasis.

METHOD.—Under local anæsthesia a hollow needle is thrust into the chest It is attached to a water manometer whose oscillations show when the pleura has been entered. Then up to 200 c c. of nitrogen are injected slowly and the needle is withdrawn. This is repeated about once a week or at longer intervals.

RESULTS —In suitable cases, i.e. when there are no pleural adhesions, the lung contracts and the cavities become smaller or are obliterated. The temperature becomes normal and the amount of sputum much less.

DANGERS.—*The production of an excessive pleural reflex* by injecting the gas before the parietal pleura has been punctured, or in cases where there are pleural adhesions which become torn by the pressure. The symptoms are pain and collapse. *Injection into the lung*, causing emphysema, and infecting the pleural cavity. Both these dangers can be avoided by only using the injection when the water manometer proves that the needle is in a free pleural cavity.

Surgery of the Chest, ***continued.***

Wounds of the Heart.—

CAUSES.—Gunshot wounds. Punctured wounds, usually of a suicidal nature.

SIGNS AND SYMPTOMS.—

1. External Signs.—Wound over precordium, with hæmorrhage or emphysema In cases in which surgical intervention is possible, external bleeding is usually insignificant.
2. Hæmopericardium —The cardiac dullness is increased, the heart-sounds are soft and distant. Venous distension in the neck, cyanosis, blueness of the lips. Marked dyspnœa. Sensation of fear and suffocation.
3. Hæmothorax.—In the worst cases the left lung and pleura are also wounded and the blood rapidly fills the chest, and death soon occurs.

OPERATION.—A large osteoplastic flap is necessary in order to gain room for manipulations It may be turned outwards or inwards, the former being the better

The flap must include the 4th, 5th, and 6th ribs and cartilages, and extend from outside the nipple line to the sternum. The costal cartilages are cut close to the sternum and the flap turned outwards by cutting and breaking the ribs at the outer margin of the flap. This exposes the pericardium over both ventricles and the left auricle If the right auricle is wounded a part of the sternum must be removed. The left lung and pleura are to be pushed back with gauze pads.

The pericardium is opened, the clots are removed, and the heart wound is sutured The flap is replaced and the wound drained

ASPIRATION OF THE PERICARDUM.—In many cases of the more favourable kind a timely aspiration of the pericardium may ward off the danger of pressure upon the heart until the wound can be dealt with.

Method —The trocar is thrust into the 5th interspace close to the sternum, inwards and backwards, and the blood removed by an aspirator

RESULTS.—In 120 cases treated by operation, 46 per cent recovered, whereas only 15 per cent of those not operated on survive. In the great majority of cases the wound affects the ventricles, the right and left being about equally involved The auricles are wounded in about 7 per cent.

CAUSE OF DEATH.—

1. Hæmorrhage.—When the wound is large, bleeding causes death before anything can be done
2. Pressure upon the Heart.—The blood collecting in the unyielding pericardium presses upon the heart, and is the cause of death in the cases of moderate severity
3. Suppuration.—In nearly 90 per cent of operation cases suppuration occurs in the pericardium or pleura, and is the cause of death in about half these.

Pericardial Effusion.—Seldom requires surgical treatment. Aspiration should be done in the 5th space, 1½ in. from the sternal margin.

Pericardial Suppuration.—May be acute from pyogenic cocci (including the pneumococcus), or chronic from tuberculous infection.

DRAINAGE is required if effusion does not yield to other measures. Drain through costosternal angle.

Direct Massage of Heart as means of Restoring Life.—In cases where the heart ceases to beat owing to the shock of trauma, operation, anæsthetic, or asphyxia, life may sometimes be restored by direct stimulation of the heart by massage or squeezing movements.

PHYSIOLOGY.—

THE EMPTYING OF A DILATED HEART.—In many of these cases the heart is distended with blood, especially on the right side. If this is expressed the muscle fibres can contract.

RESTORATION OF CORONARY CIRCULATION —The vitality of the cardiac muscle is dependent on the circulation in the coronary vessels. Massage will to some extent restore this by emptying the vessels and then allowing them to refill.

RESTORATION OF CARDIAC RHYTHM.—Rhythmical pressure on the heart walls will aid in reviving automatic rhythm of the heart.

METHODS—

SUBDIAPHRAGMATIC —A median incision is made into the abdomen through the linea alba just below the ensiform cartilage. The heart is manipulated between the diaphragm and the thoracic wall. This is the easiest, safest method, and has given the largest proportion of successes

THORACIC.—The pericardium is exposed by an incision or flap through the thoracic wall.

TRANSDIAPHRAGMATIC —The pericardium is opened through the abdomen and diaphragm. This method has had no success

ARTIFICIAL RESPIRATION must be carried on steadily irrespective of the heart manipulation.

VENESECTION —In cases with much asphyxia the withdrawal of about 12 to 20 oz. of blood will greatly relieve the tension in the heart.

RESULTS —Prospect of success depends upon following factors:—

1. THE LENGTH OF TIME during which the heart has ceased to beat, Anything longer than 5 to 10 minutes gives but little chance for restoration
2. THE CAUSE OF THE HEART FAILURE —Cases due to asphyxia or nerve shock are more likely to recover than those due to the toxic action of an anæsthetic.
3. THE NATURE OF THE CIRCULATORY FAILURE.—Cases of congestive asphyxia with cyanosis and venous engorgement give the best outlook. Cases with marked pallor are due to primary cardiac failure and have the worst prognosis But these are just the cases in which no other means are of any avail, and no time should be lost before the heart massage is begun.

Coronary Occlusion.—Is the chief cause of angina pectoris. Caused by atheroma of the coronary arteries, to which thrombosis is added.

SYMPTOMS.—Anginal attacks, dyspnœa, fatal syncope.

SURGICAL TREATMENT.—The deficient circulation in the heart can be made good by bringing a new blood-supply to it. The front surface of the heart is exposed, and a piece of the great omentum is brought up through the diaphragm and stitched to the heart.

Surgery of the Chest, ***continued.***

Cardiolysis.—In cases where the heart is hypertrophied and bound down by adhesions to the pericardium, relief may be obtained by removal of the wall of the thorax over the heart. This is termed cardiolysis.

SUITABLE CASES are those in young patients in whom rapid improvement is caused by rest, but in whom the symptoms recur on exertion.

INDICATIONS.—

SIGNS OF ADHERENT PERICARDIUM.—Especially diffuse and forcible cardiac impulse, with retraction of the precordial area on systole.

SIGNS OF CARDIAC FAILURE —Dyspnœa and œdema when standing or walking. Enlargement of the liver and spleen with ascites from venous congestion.

OPERATION.—A flap containing all the soft parts down to the ribs is turned up from the nipple to the sternum, exposing from the 3rd to the 7th cartilages on the left side. Portions of the 4th and 5th (occasionally from 3rd to 6th) ribs and cartilages are removed, about 3 to 4 in. from each. The posterior costal periosteum is left.

RESULTS —The force and volume of the pulse are improved The signs of venous congestion diminish, the liver, spleen, and œdema are lessened. The patient is relieved from distress and can do light work. About 20 cases have been reported without any fatality

Operation for Pulmonary Embolus.—It has been proposed in post-operative embolism of the pulmonary artery to expose the vessel and remove the clot (Trendelenburg).

METHOD.—An osteoplastic flap containing the 2nd, 3rd, and 4th rib cartilages of the left side is turned back and the pericardium exposed and opened. The artery is incised after the application of special hæmostatic forceps and the clot extracted piecemeal. The vessel is then sutured

RESULTS —About eleven successful cases have been reported. In others, death has occurred at the time or shortly afterwards, usually from septic or pulmonary complications

CHAPTER XXXIII

DISEASES OF THE BREAST

Ulcers of the Nipple.—

SIMPLE CRACKS AND FISSURES.—Caused by lactation. Result often in abscess in the breast.

ECZEMA.—

1. Ordinary acute eczema, recovering under treatment.
2. Chronic eczema, with much scaling, but no destruction of the nipple or induration.
3. *Paget's Disease.*—A form of chronic eczema The nipple becomes destroyed. The deep surface of the eczematous patch is hard and indurated. Carcinoma of the underlying breast is associated with it. This is usually regarded as resulting from the nipple condition. But it may be the primary disease and cause destruction of the nipple by destroying and permeating the lymphatics (Handley).

TREATMENT —Remove whole breast as for carcinoma if no improvement

SYPHILIS.—(1) Primary chance, rare in the mother of a syphilitic child. Axillary glands large and hard (2) Secondary mucous patches. (3) Tertiary gummata and ulcers, very rare.

EPITHELIOMA.—Crater-like ulcer with hard everted edges. Much destruction of skin and nipple.

SCIRRHUS OR SARCOMA.

Malformations.—The chief are supernumerary breasts and hypertrophy.

HYPERTROPHY occurs in young women, apart from pregnancy and lactation; causes fatigue and distress.

TREATMENT —Plastic operation, with preservation of nipple and removal of redundant tissue

INFLAMMATORY DISEASES OF THE BREAST

Acute Mastitis : Acute Mammary Abscess.—

CAUSES.—Infection during lactation, either by the ducts or the lymphatics. Pyæmia. New-born infants, generally after injury, e g., 'breaking the nipple-string'. Extension from other structures, e g., pleura or ribs.

VARIETIES.—

SUPRAMAMMARY.—Abscess forms between the skin and breast.

Treatment.—Simple incision rapidly cures it.

INTRAMAMMARY —Abscess forms in the breast substance. At first is limited to one or more lobules. It is partly limited by fibrous radiating septa. The whole breast swells because of retained milk.

Treatment.—By free radiating incision; break down septa in the cavity so that no 'pockets' remain. Counter-opening low down for drain.

SUBMAMMARY.—Between breast and chest wall. May arise from rib or chest disease. Breast is pushed forward, but not inflamed.

Treatment.—By opening below and to the outer side.

Inflammatory Diseases of the Breast, ***continued.***

Chronic Mastitis.—

1. LOBAR.—Remains after lactation, from imperfect involution of one or more lobes. Results from injury or an acute mastitis.
   SYMPTOMS.—Pain of neuralgic character. Worse during menstrual period.
   DIAGNOSIS from cancer. Flat hand feels no tumour. No craggy edge.
   TREATMENT.—Rest. Firm bandage. Belladonna.
2. LOBULAR OR INTERSTITIAL.—Women about the menopause. Often in thin and sterile women.
   PATHOLOGY.—(*a*) Overgrowth of interstitial fibrous tissue; (*b*) Contraction of this fibrous tissue; (*c*) Consequent pressure on ducts and acini; (*d*) Epithelial proliferation, (*e*) Dilatation of acini to form cysts. Cysts are filled with thick, dark, mucoid fluid. There are no intracystic growths
   SIGNS.—Nodular feeling in the whole breast. One or more lumps can be seen and felt The flat hand often feels nothing against the chest wall Lymph-glands are often a little large and tender. Nipple may be retracted Often present in both breasts. Pain is inconstant, and is worse at menstruation Definite cysts form elastic swellings. Often history related to trauma
   TERMINATIONS.—Atrophy—Polycystic or fibrocystic disease—Carcinoma.
   DIAGNOSIS.—From cancer or adenoma.
   TREATMENT.—Short period of firm pressure and application of X rays, and then excision if improvement has not occurred. Hormone therapy, e g, stilboestrol
3. FAT NECROSIS.—Is really a form of local mastitis. Occurs in middle-aged women. Usually results from trauma, e g, a blow or the insertion of a transfusion needle An irregular nodule with dimpled skin, exactly simulating a scirrhus. Consists of lobules of necrosed fat, surrounded by area of fibrous tissue reaction
   TREATMENT.—Excision after biopsy.

Chronic Abscess, Non-tuberculous.—May be the result of low-grade infection in a hæmatoma, in an imperfectly resolved acute mastitis. Slow painful tender swelling of breast, which is enlarged and pushed forward. Thick-walled abscess is formed.

TREATMENT.—Opening, draining, or dissecting away the abscess walls completely, penicillin is of value in these cases.

Tuberculosis of the Breast.—Rare; 20–35; unilateral in 60 per cent cases; secondary to known infection elsewhere in lungs and glands. Fibrosis with sinus formation and typical tuberculous granulation tissue.

TREATMENT.—Removal of breast and glands, and treat as for tubercle by sanatorium treatment.

CYSTS OF THE BREAST

RETENTION CYSTS.—From the dilatation of acini. Often have serious discharge from nipple.

GALACTOCELE.—Filled with altered milk. Arises during or after lactation.

DISTENSION CYSTS.—

INVOLUTION CYSTS.—Occur in interstitial mastitis blocking the ducts. Caused by contracting fibrous tissue.

IRRITATION CYSTS.—From irritation of nipple, causing reflex secretion.

INTERACINOUS CYSTS.—
Serous Cysts.—Dilated lymph space. Lined by endothelium. Never gives rise to discharge from nipple.
Hydatid Cysts.
TUMOUR CYSTS (*Fig.* 148).—
Adenoma
Duct Papilloma } Cysts have intracystic growths, and often a serous or bloody discharge from the nipple.
Duct Carcinoma
Carcinoma } From hæmorrhage or degeneration.
Sarcoma

SIMPLE TUMOURS OF THE BREAST

VARIETIES.—Fibro-adenoma—Adenoma—Lipoma and chondroma (very rare)—Duct papilloma.

Adenoma Mammæ.—

CONSISTS of tissue like normal breast-gland tissue
1. Firm Fibrous Capsule enclosing—
2. Epithelial Alveoli —Generally in a single layer—Without ducts —Often dilated to form cysts—Cysts often contain intracystic growths.
3. Interstitial Fibrous Tissue.

VARIETIES —
Pure Adenoma, or acinous adenoma —Structure similar to the normal gland
Fibro-adenoma, or tubular adenoma —Much the commonest Fibrous tissue is out of proportion to glandular Alveoli are drawn out into long canaliculi A fibrous ingrowth takes place into these canaliculi.
Cysto-adenoma, or serocystic disease —Alveoli are dilated into cysts. Filled with fibro-papillomatous proliferations. Generally form a large tumour, which may burst through the skin Often becomes sarcomatous.

SIGNS.—
Age —From puberty to thirty is the time of their origin.
Cause —Often follow a blow
Pain.—Neuralgic, worse at menstruation.
Tumour.—Hard, oval, and elastic Freely movable in breast substance. Not fixed to skin, nipple, or chest Does not produce enlargement of axillary glands. Often multiple and in both breasts On removal section is hard, white, foliated, and encapsuled No juice can be scraped from it.

TERMINATIONS.—Remain unaltered Become carcinomatous or sarcomatous

TREATMENT.—Removal.

Duct Papilloma.—Small tumour near the surface and near the nipple (*Fig.* 148). An epithelial papilloma growing from the lining of a duct. A cyst about the size of a cherry, containing a warty growth. Serous or bloodstained discharge from the nipple Growth is not indurated, and is sharply defined. No enlarged glands.

Becomes a duct carcinoma by the epithelium of the papilloma growing down through the wall of the duct

TREAT by removal of the breast.

Duct Carcinoma.—Similar to the above, but tumour has an indurated base. Lymph-glands enlarge. Less rapid and malignant than ordinary cancer.
TREAT by removal of the breast.

MALIGNANT DISEASE OF THE BREAST

Carcinoma.—

VARIETIES.—

1. Arising From Alveoli.—Spheroidal- or polygonal-celled
2. Arising From Ducts.—Columnar-celled.
3. Arising In The Skin Of The Nipple.—Squamous-celled.

Sarcoma.

SPHEROIDAL-CELLED CARCINOMA

This group comprises: (*a*) Atrophic scirrhus: (*b*) Acute scirrhus; (*c*) Encephaloid.

Aetiology.—The proportion of females to males is 100 to 1. More common site for cancer than any other organ except the uterus.

Liability is due to: (1) Rapid structural changes accompanying puberty, lactation, and menopause; (2) Irritation of lactation; (3) Exposure to injury

May follow: Eczema (Paget's disease)—Mastitis—Adenoma.

Age forty to fifty, at about the menopause.

Course.—Lasts from about one to three years. In old women may be a chronic disease and last fifteen years In young people is a rapid disease.

Anatomy of Scirrhus Mammæ.—Hard mass in the breast, looking like white fibrous tissue. Tumour is generally small and the breast contracted. Craggy edge with no capsule Cut surface is concave, and yields juice on scraping It cuts with a grating sensation Is ill defined from surrounding structures It infiltrates breast, connective tissue, muscles, and chest wall, the tissues becoming actually replaced by cancer cells. (*See Fig.* 23, p 67)

MICROSCOPICALLY—Columns of spheroidal cells—the cancer cells Generally three or four rows of cells in each column. Columns divide and branch irregularly. Quantity of fibrous tissue surrounds the column of epithelial cells

Mode of Extension.—

1. DIRECT INVASION OF THE ADJACENT STRUCTURES—The pectoral fascia, pectoral muscles, skin, chest wall, or pleura may be involved by extension of the primary growth
2. METASTASIS, OR THE EXTENSION TO DISTANT STRUCTURES.—

Permeation of the Lymphatics, in which the cancer cells grow along the lymph-vessels in direct continuity with the growth, is the mode in which this probably occurs.

Perilymphatic Fibrosis then takes place on the rupture of those lymph paths which have been first affected, and the cancer cells disappear by this process in the central zone.

The Microscopical Growing Edge of the cancer cells can be demonstrated in the lymph-vessels of the deep fascia, disposed like a circle round the primary growth, the diameter of the circle enlarging with the age of the case.

Metastases occur at any point where the cancer cells have permeated, by a side path or in their direct line of growth, some neighbouring structure, or where perilymphatic fibrosis has failed.

The Lymph-glands are naturally the first and most important seat of secondary growth: (*a*) In the axilla, the pectoral, apical, and subscapular sets are early involved; (*b*) In the neck, the glands in the posterior triangle are infected by extension from the axillary set; (*c*) In the mediastinum, the glands may be infected by the lymph-vessels which accompany the perforating branches of the internal mammary, or by extension from the neck.

Skin Nodules may thus occur in an area which is roughly circular and which has the primary growth as its centre. They never attack the distal parts of the limbs, and are rare except on the trunk, neck, and head.

Bone Metastasis, too, occurs with a frequency for individual bones which is in proportion to their proximity to the growth, and chiefly at such points as the deltoid insertion on the humerus, or the great trochanter of the femur, where the lymph-vessels of the deep fascia are continuous with the periosteum. The bones most commonly affected are the sternum, clavicle, humerus, ribs, and vertebræ.

Trans-cœlomic Implantation is the process by which the cancer cells, when they have reached the pleura or peritoneum, may fall free in these cavities, and cause growths on the surface of the contained viscera.

Symptoms and Signs.—There are always three stages :—

1. STAGE OF EARLY GROWTH WITH DOUBTFUL CHARACTERS. —Hard nodule in the breast, best felt between the flat hand and the chest Quite painless, and only noticed when washing. No tenderness. Generally in an outlying lobule of the breast. It is incorporated with the gland substance, but at this stage is fixed to nothing else. It steadily increases in size Usually in upper and outer quadrant of breast.

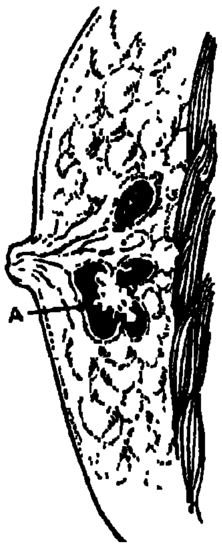


Fig. 148.—Cysts in the breast. A, Duct papilloma.

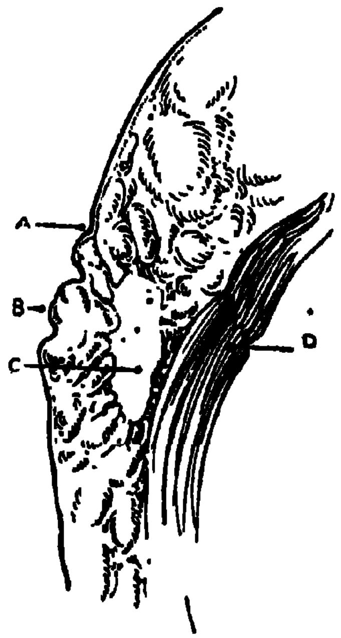


Fig 149.—Scirrhous carcinoma of breast. A, Skin drawn in by contracting bands of the growth; B, Retracted nipple; C, Growth; D, Pectoral muscle (not affected).

Spheroidal-Celled Carcinoma—Symptoms and Signs, ***continued.***

2. STAGE OF ESTABLISHED GROWTH WITH WELL-DEFINED CHARACTERS —

HARD GROWTH, which has a nodular, craggy, ill-defined margin.

FIXED to the suspensory ligaments, joining it to the skin, producing dimpling.

To the skin itself later on, when it may ulcerate.

To the nipple, dragging on its ducts and producing retraction (*Fig.* 149).

To the pectoral muscle, fixing the breast so that it cannot be moved up and down the length of the muscle fibres (*see Fig.* 23, p. 67).

To the chest wall, so that the breast cannot be moved across the direction of the muscle fibres

LYMPH-GLANDS form hard, fixed, nodular mass in the axilla and posterior triangle of neck

PAIN is marked in proportion to fixation. Is neuralgic and intermittent.

SKIN.—May be affected in one of several ways:—

1. Brawny, congested, and fixed to the growth.
2. Ulcerated. Smooth, foul ulcer, whose base is formed by the growth
3. Nodules of secondary growth over the breast, or at a distance on the skin of any part of the trunk
4. Cancer *en cuirasse*, the skin forming a layer of cancer-invaded tissue. The 'orange skin' or 'pig skin' often precedes this, the mouths of the glands being opened and large with excess of secretion.
5. Cancerous lymphangitis The lymph-vessels are filled with cancer cells, and lymph exudes from the lymph capillaries, giving an appearance of weeping eczema

DISCHARGE from nipple—blood-stained—is only occasionally present.

3. STAGE OF CACHEXIA —Rapid emaciation. Marked anæmia Skin becomes loose and inelastic all over the body. Growth is densely fixed or widely ulcerated Lymph-glands by pressure on the vessels and nerves cause œdema and pain in the arm Solid brawny œdema of the arm due to the wide destruction of lymphatics by perilymphatic fibrosis. Pleurisy or ascites may occur from metastatic growths Pain constant and unbearable

Diagnosis.—In Stage 1 the diagnosis is always conjectural until the growth is under the microscope; but until proved innocent every doubtful tumour should be regarded as malignant

FROM MASTITIS.—In this the flat hand against the breast can define no tumour. The development is after a blow or lactation. The edge is not hard or craggy. Generally tender. There may be retraction of skin and nipple and enlargement of the lymph-glands.

FROM FIBRO-ADENOMA.—In this the tumour floats about in the breast substance Begins before thirty, generally about twenty. It gives pain of shooting kind, worse at menstruation. Very definite outline.

FROM A CYST.—In this there may be other signs of mastitis. Swelling is elastic or fluctuating. History is a long one

FROM A CHRONIC ABSCESS OR TUBERCULOUS FOCUS.—In this the history is a long one. Pain or tenderness are early. Centre of swelling is softer than the margins.

FROM SWELLINGS OF THE CHEST WALL, e.g., tubercle or actinomycosis of chest wall, aneurysm, etc. These are obviously fixed to the chest, whilst the breast moves over them.

ENCEPHALOID CARCINOMA

Differs from scirrhus in the following points:—

Anatomy.—Cancer cells are larger. Columns of cancer cells are thicker. Interstitial fibrous tissue is scanty. Growth is large, soft, and vascular.

Aetiology.—Occurs in young women, twenty-five to thirty-five. Often follows lactation or pregnancy.

Course.—Very rapid, ending in about six months.

Signs.—Soft or elastic tumour of some size. Very rapid growth. Often feels hot from its vascularity. Is painful in its early stages Lymph-glands are affected rapidly. Skin is broken through by a fungating mass. Retraction of skin and nipple are absent.

Treatment.—Preliminary treatment by radiotherapy will frequently reduce the tumour in size and make it more amenable to radical treatment.

Diagnosis is never long in doubt.—

FROM ACUTE MASTITIS —In this an abscess forms, upon opening which the swelling and signs subside. All the signs of inflammation. High temperature

FROM SARCOMA —In this the tumour is round and circumscribed. Pain is absent. Lymph-glands are not involved early.

OTHER VARIETIES OF CARCINOMA MAMMÆ

Atrophic Scirrhus.—Women over sixty. Very slow in course, lasting ten to fifteen years. Great excess of fibrous tissue. Very scanty cancer cells. Produces atrophy of breast.

Colloid Carcinoma.—A degeneration of any spheroidal-celled carcinoma. Cancer cells become colloidal.

MICROSCOPICALLY an irregular network of interstitial tissue contains a few degenerate cells and large clear spaces where the colloid takes no stain.

SIGNS.—Enlargement more rapid. Softer character. Does not lessen the rapidity of the disease.

Acute Cancer or **Lactation Carcinoma.**—Even more acute than the encephaloid type May be mistaken for acute mastitis. Differentiated by absence of pain. Treatment is hopeless Ends fatally in six weeks to three months.

SARCOMA MAMMÆ

Varieties.—

ROUND-CELLED.—Like lymph tissue. Very rapid. Many metatases, especially in lungs. Lymph-glands seldom involved.

Sarcoma Mammæ—Varieties, *continued.*

SPINDLE-CELLED.—The common variety. Cells are oat-shaped. Slower in growth. Definitely encapsuled at first.

Aetiology.—Any age, without reference to functional changes. Old women and young are often subject to it. No relation to lactation. May follow fibro- or cysto-adenoma.

Signs.—

TUMOUR is elastic or semi-fluctuating. Round and well defined. Very rapid growth.

SPREAD.—Skin is first involved. A fungating, bleeding mass protrudes. Deep tissues are invaded later.

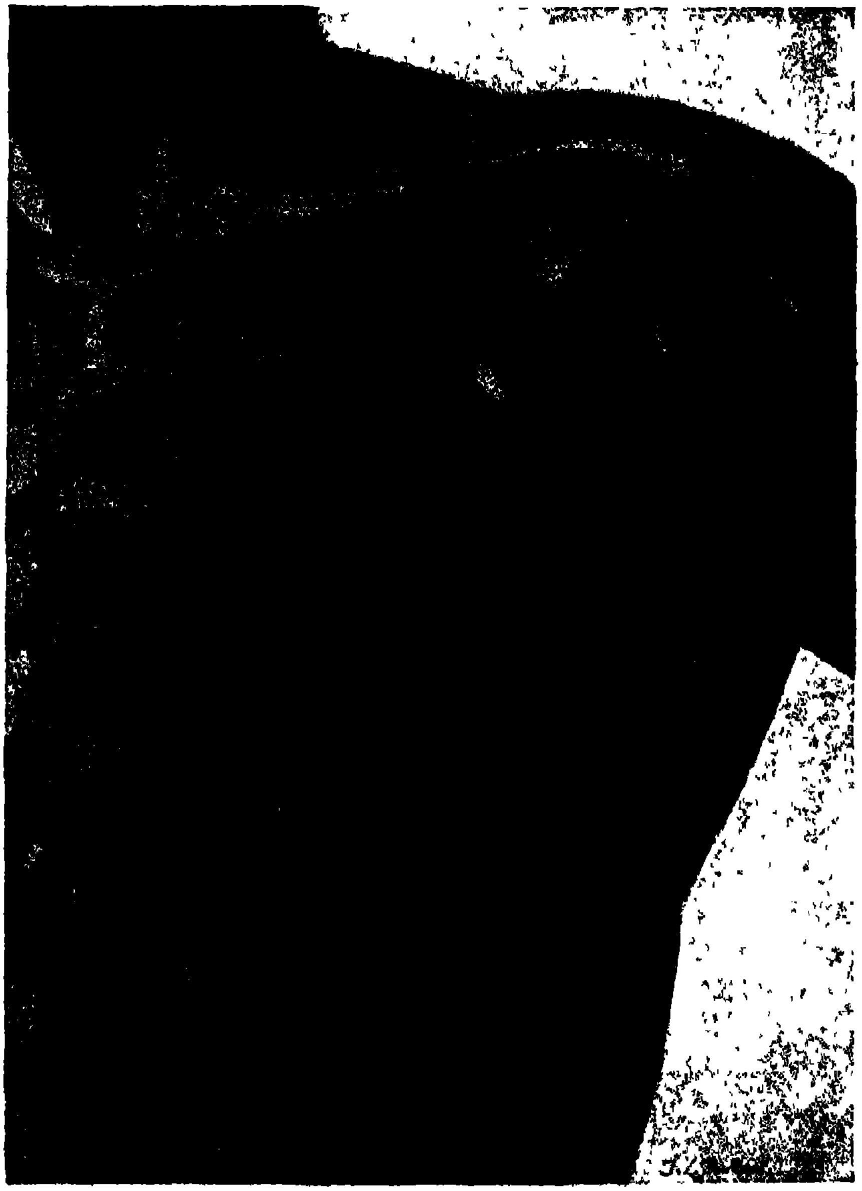


Fig. 150.—Diagram showing disposition of radium needles.

METASTASES.—Lymph-glands, especially in spindle variety. Lungs and pleura.

DEGENERATIONS.—Hæmorrhage—Spurious cysts—Myxomatous degeneration.

TREATMENT OF MALIGNANT DISEASE OF BREAST

1. RADICAL REMOVAL always involves:—
   a. Removal of whole gland with a good margin.
   b Removal of wide area of skin over breast—4–6 in., with nipple at centre.
   c. Removal of pectoral fascia: better with the sternocostal part of muscle.
   d. Removal of whole of axillary glands, pectoral, subscapular, and apical groups.

   Occasionally: removal of pectoralis minor in order to clear glands higher, removal of supraclavicular glands

   SPECIAL POINTS.—

   Cases of doubtful diagnosis: excise tumour, and microscope, without waiting for typical signs.

   Cases where much skin is removed (*a*) Undercutting edges of skin; (*b*) Cutting skin flaps, (*c*) Thiersch's skin-grafting

2 CASES UNSUITED FOR RADICAL OPERATION —

   Skin conditions: Cancer *en cuirasse*—Widely diffused nodules of cancer —Cancerous lymphangitis

   Adhesions to chest wall

   Lymph-glands causing œdema of arm, or forming a mass in posterior triangle of neck

   Metastases: Pleura, peritoneum, or bones

   Cachexia, with rapid wasting

3. METHODS OF TREATMENT FOR INOPERABLE CASES —

   Radium needles or radon seeds (*see Fig* 150)

   Keynes has shown that the lymphatics of the breast tend to run through the breast tissue and deep structures or muscles into the axilla rather than along the fascial planes In this case wide removal is necessary, and radium with or without removal of the breast is of great value. Some surgeons are treating carcinoma of breast by radium only with satisfactory results

   X-ray treatment: For recurrent nodules in the skin Lessens pain and fixation. Lengthens life

   For the brawny œdema of arm Insertion of subcutaneous silk threads from wrist to chest Act as capillary drains (Sampson Handley).

CHAPTER XXXIV

INJURIES OF THE ABDOMEN. PERITONITIS

AFFECTIONS OF THE ABDOMINAL WALL

Rupture or Hernia is considered in Chap. XXXVIII.

Rupture of Muscle or Hæmatoma.—May result from violent strain, a blow, or tetanus. Usually affects the rectus. Muscle is torn and blood collects in the sheath.

TREATMENT.—Evacuate blood-clot and repair the muscle by suture.

Neoplasms.—Sarcoma or epithelioma may occur.

DESMOID TUMOUR, the recurrent tumour of Paget, is a rare tumour of the sheath of the rectus. It is a cellular, unencapsuled fibroma which recurs unless completely removed.

TREATMENT —Complete removal

KANGRI CANCER —Squamous-cell cancer seen in people of Kashmir.

Never in the midline; 75 per cent cases below the umbilicus; commoner in females than in males.

Painless, with skin moving freely over it.

TREATMENT —Excision:

CONTUSIONS OF THE ABDOMEN

Results.—

PARIETAL.—Contusion of abdominal wall—If infected, abscess in the abdominal wall—Rupture of abdominal muscle—Subsequent hernia—Rupture of parietal peritoneum—Bleeding from vessel in parietes, from vessel in abdomen

VISCERAL.—

RUPTURE OR CONTUSION OF VISCERA — Liver — Spleen — Kidney — Pancreas—Bladder—Stomach—Intestine—Pregnant uterus—Ovarian cyst.

PERITONITIS, or PERITONEAL EXTRAVASATION of stomach or bowel contents—Gas—Blood—Urine—Pancreatic juice

Symptoms.—

FIRST STAGE · SHOCK.—Pallor, sweating, thirst, subnormal temperature, faintness, rapid weak pulse Evidence of local injury.

SECOND STAGE: RECOVERY FROM SHOCK.—Pulse and temperature may be normal and general symptoms absent, yet severe visceral lesions may be present. If present, third stage soon follows.

THIRD STAGE: ONSET OF COMPLICATIONS.—

1 INJURY OF SOLID VISCUS.—Liver, spleen, kidney, pancreas. Symptoms are due to internal hæmorrhage These are. gradually increasing pulse-rate, also becoming weaker, and all symptoms given under shock above, but in addition restlessness and increased respiration, which may be sighing. These latter two symptoms are important in differentiating internal hæmorrhage from pure shock. The abdomen becomes distended, and there is shifting dullness in the flanks,

2. Injury of Hollow Viscus.—**Stomach or intestines. Resulting symptoms are those of peritonitis. Persistent vomiting, rising pulse-rate, distension of abdomen by tympanites, loss of liver dullness. Increasing tenderness and rigidity of abdominal wall.**
3. Injury to Bladder.—**Inability to micturate, or passage of small quantities of blood and urine. There is often shifting dullness, and later the above signs of peritonitis.**

Diagnosis.—Early diagnosis is usually difficult, though essential for successful treatment. Radiography may be useful, in showing free gas in the abdominal cavity. If visceral injury is suspected, withhold morphia and food by mouth, give rest and warmth, take the pulse every hour, and if, after recovery from initial shock, the pulse-rate rises and any of above symptoms increase, treat as for visceral injury. It is far better to look and see than wait until signs are markedly obvious.

Treatment of Visceral Injury.—Immediate exploratory laparotomy, dealing with such injury as is found. When in doubt, operate, is the safe rule Delay is dangerous. Within 4 hours of injury, mortality is only 15 per cent. After 12 hours, mortality is 70 per cent Prepare for blood transfusion. Special anaesthesia, e g., spinal, local, or gas and oxygen.

WOUNDS OF THE ABDOMEN

NON-PENETRATING.—This character can only be properly ascertained by carefully excising the wound as a whole down to its bottom. If the peritoneum is uninjured, the other layers are sutured seriatim. Blind probing should always be avoided, as liable to cause penetration and sepsis.

SMALL PENETRATING WOUNDS, e.g., stabs, etc —The escape of blood, fæces, gas, bile, or urine will establish the fact of visceral penetration. Later there will be signs of peritonitis, but all these may be absent; the wound should nevertheless be opened up without delay and the underlying viscera examined before infection has had time to be established

MULTIPLE WOUNDS.—Here a median incision is usually necessary to deal with the visceral injuries, whilst the wounds are excised and separately closed.

LARGE WOUNDS —In these there is great intestinal protrusion. The protruded viscera and the abdominal wall must be carefully cleansed before reduction Close the parietal wound in at least three layers.

GUNSHOT WOUNDS.—Gunshot wounds ought always to be operated upon. If penetration is doubtful, radiography and opening the track of the bullet will determine this point. If penetration is obvious, and especially if there are multiple wounds, a large median incision is required, and the viscera must be systematically examined and sutured where necessary.

PERITONITIS

Peritonitis may be acute or chronic, and localized or generalized.

Causes.—

1. FROM WITHOUT.—Penetrating wounds—Puerperal peritonitis.
2. FROM STOMACH OR INTESTINES.—Injury, ulcer, or perforation.
3. FROM FALLOPIAN TUBES.—Gonococcal—Pneumococcal.
4. FROM BLOOD — Tuberculous — Staphylococcal — Streptococcal — Pneumococcal.

Peritonitis, *continued.*

Bacteriology.—

Bacteria inhabiting the intestinal canal play an important part in peritonitis. Peritoneal infection is invariably a mixed one, but the following groups of organisms are found: (1) AEROBES: *B coli*, *B. pyocyaneus*, *B. proteus*, and pyogenic cocci, as *Staph. pyogenes*, pneumococci, and *B. tuberculosis*; (2) ANAEROBES: *B. welchii*.

ACUTE GENERAL PERITONITIS

Anatomy.—

DILATATION OF VESSELS lying beneath peritoneum Best seen in the coils of intestine.

EXUDATION of lymph upon peritoneal surface, which becomes sticky. This is seen first in the angles between adjacent coils of intestine. Exudation of serum and leucocytes and bacteria to form purulent exudate. This falls into the most dependent parts—loins and pelvis

SMALL ROUND-CELLED EXUDATION beneath the peritoneum.

ENDOTHELIUM IS SHED in virulent infections In this case, if recovery occurs, the lymph is organized by the round-celled layer, and forms permanent fibrous adhesions

If the endothelium is not shed and recovery occurs, the lymph adhesions are absorbed

ADHESIONS —First lymph, and later organized granulation tissue (*see above*), mat together adjacent viscera. The omentum especially adheres to the focus of greatest inflammation

Symptoms.—

GENERAL.—Profound collapse—Drawn face—Small, rapid, wiry pulse —Quick and shallow respirations—Temperature variable, low in worst cases—Vomiting (little force, dark or fæcal)—Hiccup—Constipation

LOCAL.—Extreme tenderness—Rigidity of the abdomen very marked—Pain local and then diffuse—Breathing purely thoracic—Tympanites —Legs drawn up—Dullness in flanks after two or three days.

Differential Diagnosis.—

1. THORACIC LESIONS.—Pleurisy, pneumonia, coronary thrombosis may give symptoms and signs referred to the upper abdomen, and may be mistaken for acute cholecystitis and perforation
2. ABDOMINAL COLIC —Biliary and renal.
3. INTESTINAL OBSTRUCTION.—This may lead to peritonitis, but in early stages there is absence of tenderness and rigidity and exaggerated intestinal sounds.
4. INTRAPERITONEAL HÆMORRHAGE.—Trauma.
5. RENAL DISEASE.—Pyelitis may be misleading
6. SPINAL CORD AND COLUMN.—Tabes dorsalis and gastric crises.
7. TORSION OF OVARIAN CYST.

Treatment.—

GENERAL.—Rest and heat to abdomen, e.g., electric pad. The lower bowel should be cleared by a simple or turpentine enema, but it is useless to repeat this. The stomach should be washed out at regular intervals, and this is best done by keeping a Rehfuss tube in the stomach if possible. Avoid purgatives. Morphine will almost certainly have to be given for pain. If marked distension exists, the relief of this is imperative. Acetylcholine or carbochol and flatus tube. Prostigmin (1 c.c.) every hour for 6 hours.

CONTINUOUS SALINE INFUSION BY RECTUM.

SALINE BY INTRAVENOUS INJECTION.—Of great value because it makes good the chloride deficiency which has resulted from the vomiting.

ANTI-GAS-GANGRENE SERUM (*B. welchii*).—Give 20 to 30 c.c. intravenously. This helps to control the toxæmia, which is probably due to anaerobic organisms in the distended and paralysed gut. Of little use in attacking the ileus that is present.

HUMAN OR OX BILE —Administered per rectum this relieves the incessant vomiting associated with general peritonitis.

OPERATIVE.—

OPEN ABDOMEN: over seat of injury if known; in mid-line if not known.

SEEK CAUSE of peritonitis and deal with it.

EMPTY ALL FREE FLUID by sponging, and manipulate the peritoneal surfaces as little as possible.

If a local peritonitis only is present, e g., in the great majority of cases of appendicitis.

SUMMARY.—(1) Removal of cause. (2) Peritoneal toilet. (3) Drainage. (4) After-treatment, as replacing fluid and chloride loss, and the treatment of paralytic ileus if present.

LOCAL PERITONITIS

Cause.—Abdominal injury, or visceral injuries, with infection of low degree of virulence.

Varieties.—Abdominal contusion—Subphrenic abscess (*see* p. 386)—Leaking gastric ulcer or carcinoma—Cholecystitis, or leaking gall-bladder—Duodenal or other intestinal ulcer—Appendicitis— Pelvic peritonitis—Inflammation of ovarian tumour or of uterine appendages—Inflammation of appendices epiploicæ.

Symptoms.—

1. LOCAL PAIN AND TENDERNESS.—Symptoms referable to organ involved, e.g., dyspepsia, jaundice, uterine discharge
2. LOCAL SWELLING.—Induration Dullness to percussion. Increase of visceral symptoms.
3. SIGNS OF LOCAL ABSCESS —Raised and remittent temperature. Fluctuating or œdematous swelling. Abscess bursting internally or externally.

Treatment.—First, rest and heat, and then operate—Remove cause.

PNEUMOCOCCAL PERITONITIS

Primary or secondary. Much commoner in children than adults.

Primary Form.—Either a blood infection or else an infection through the Fallopian tubes. In support of the latter theory: It is rarely found except in dirty ill-cared-for children, usually under 12 years of age; it is almost confined to the female sex; pus shows specific organisms.

Pneumococcal Peritonitis—Primary Form, *continued.*

CHARACTERISTICS.—A very acute form of peritonitis which runs to a fatal course within two to seven days, if not treated.

Diarrhœa, vomiting, and painful micturition are all marked as early symptoms. The signs begin as rigidity of lower abdomen. Child obviously gravely ill. Vaginal discharge is usually present in children.

Secondary Form.—Occurs with equal frequency in both sexes in association with pneumonia. It affects the whole peritoneum uniformly. It is often overlooked by reason of the existence of the chest condition.

Treatment.—Early laparatomy with evacuation of the thick greenish purulent exudate and drainage.

Transfusion of citrated blood is a powerful adjuvant to recovery. Anti-pneumococcal serum and chemotherapy.

CHRONIC PERITONITIS

Causes and Varieties.—(1) Simple irritation, or septic infection of a mild type; (2) Tubercle; (3) Malignant disease.

Simple Chronic Peritonitis is of two different types:—

1. LOCAL FIBROUS PERITONITIS.—The result of septic invasion which has been of a mild character. It may accompany, or remain after, any acute local infection, and is therefore common round gall-bladder, stomach, appendix, or uterine appendages. May occur round a sterile foreign body which has been left in the abdomen.
2. SIMPLE PERITONITIS OF UNKNOWN ORIGIN.—In rare instances general chronic peritonitis arises, with effusion and thickening of the peritoneum, but with no clear evidence of its cause. Its association with RHEUMATIC FEVER and BRIGHT'S DISEASE suggests that it is toxic in origin. The peritoneum, especially over the liver and in the great omentum, becomes much thickened and distorted, and there is a marked ascites.

Tuberculous Peritonitis is most commonly found in children and young adults.

ORIGIN.—

1. As a diffuse miliary invasion of the peritoneum, the tubercles occurring in the course of the blood-vessels.
2. From the intestine, either appendix, ileum, or cæcum, and other parts more rarely. In this case the lymph tissue of the gut becomes infected, and thence the disease spreads to the peritoneum.
3. From the uterine appendages: this being one of the commonest modes of origin in women.
4. From tuberculous glands in the mesentery.

VARIETIES.—(1) Ascitic; (2) Fibrous; (3) Suppurative, or ulcerous; (4) Encysted or loculated.

THE ASCITIC VARIETY.—This is, perhaps, the commonest, and certainly the most mild variety.

SIGNS.—The usual signs of distension with free fluid are present. There is a little thickening of the peritoneum and mesentery, but adhesions are absent. The fluid is straw-coloured, highly albuminous, and contains many leucocytes. Omentum is rolled up and feels like a band across the abdomen. Effusion is slow in onset, with progressive ill health and loss of vitality. Abdomen is distended, skin tense and shiny, with dilated veins on it. Abdomen is doughy, TREATMENT: (1) Constitutional. (2) Laparotomy without irrigation or drainage. The slight beneficent effect is probably due to the outpouring of serum, containing antibodies, after the removal of the fluid. Has little to recommend it, and only when the distension is very great and causing embarrassment.

THE FIBROUS VARIETY.—

ANATOMY.—There is marked thickening of the mesentery and omentum, the latter often being rolled up as a dense band below the colon. Adhesions form by which the intestines become matted together, especially round the primary focus of disease. Contraction of the mesentery also tends to produce kinking and, together with the adhesions, may cause intestinal obstruction. In the later stages fistulæ occur.

THE PHYSICAL SIGNS are those of some free fluid, and also irregular masses can be felt in the abdomen in the situation of the groups of adhesions The lymph-glands in the mesentery and behind the peritoneum are notably enlarged.

THE TREATMENT is usually only constitutional; the result of operations being as a rule very bad, owing to the ready way in which the diseased intestine is torn, and the impossibility of removing the affected tissues.

THE SUPPURATIVE OR ULCEROUS VARIETY.—

ANATOMY.—This is a further development of the last variety. In the midst of the masses of adherent gut and omentum, foci of caseation and suppuration occur. It is common round the tuberculous uterine appendages. The bowel itself is often the seat of advanced tuberculous ulceration. Secondary infection of any such suppurating foci by the intestinal bacteria is common.

PHYSICAL SIGNS are similar to the last case, but localized fluctuating swellings may make their appearance, or break through the parietes, especially at the navel.

SYMPTOMS.—These are as insidious and ill-defined as in the case of tuberculous lesions elsewhere. Some hectic fever is present in acute or late cases. Emaciation, loss of appetite, and indefinite abdominal pain. Intestinal obstruction is common in the adhesive and suppurative types.

TREATMENT.—By laparotomy when constitutional treatment has failed. An attempt should be made to remove the primary focus of the disease when this is in the Fallopian tube, appendix, or lymph-glands. There is a grave risk of wounding the bowel and leaving a fæcal fistula.

THE ENCYSTED OR LOCULATED TYPE.—Frequently in young women. Definite tumour with matting together of coils of intestine with a loculated cavity containing fluid. Tubercles and thickened peritoneum surround the area, but the rest of the peritoneum is healthy. If untreated, condition deteriorates. Sanatorium treatment, and if no response, operation should be performed.

Malignant Peritonitis.—

CAUSES.—

1. Primary malignant disease of the peritoneum. This is an endothelioma, and begins as a rule in the pelvis.
2. Secondary deposits from papilliferous cysts, especially those growing from the hilum of the ovary. These burst through the capsule of the primary growth and become implanted all over the peritoneal surface.
3. Deposits secondary to visceral carcinoma, especially of the stomach, liver, and intestine.

Malignant Peritonitis, ***continued.***

PHYSICAL SIGNS.—Pain, emaciation, and the presence of some tumour, precede the collection of free abdominal fluid.

THE FLUID WITHDRAWN is usually blood-stained, but in the case of the ovarian papilliferous cysts it is thick and mucoid.

CHRONIC PERITONEAL EFFUSIONS: ASCITES

CAUSES.—

1. Chronic peritonitis, tubercle, new growth, etc.
2. Cardiac and renal disease.
3. Portal obstruction. Cirrhosis of the liver Malignant disease in the liver or portal glands.

PHYSICAL SIGNS.—

BARREL-SHAPED DISTENSION OF THE ABDOMEN, the skin becoming shiny and marked by 'striæ'.

PERCUSSION shows areas of dullness in the flanks during recumbency, in the hypogastrium in the erect posture, or all over if distension is so great that the mesentery is too short to allow the gut to float against the parietes. In typical cases the areas of dullness become resonant when the patient turns over.

FLUID THRILL is felt on one side of the abdomen when the other is percussed: distinct in proportion to the tension of the fluid.

THE FLUID derived by tapping is clear and almost colourless, and contains little albumin and few blood-cells in the obstructive cases as compared with the inflammatory or malignant.

ANATOMICAL VARIETIES.—

1. DIFFUSE.—In this, by far the commoner variety, the fluid occupies the great sac of the peritoneum.
2. LOCALIZED.—(*a*) In the lesser sac. Often arising from pancreatic disease, and giving the signs of pancreatic cyst. (*b*) In the pelvis, especially in women, when it resembles a retroperitoneal or broad ligament cyst. (*c*) In the omentum or mesentery, when it resembles a mesenteric cyst.

TREATMENT.—

TAPPING by a trocar and cannula over a dull area. This is done most effectively and with least discomfort to the patient by Southey's capillary tubes, one being introduced into each flank, and allowed to drain for several hours.

EPIPLOPEXY, or TALMA'S OPERATION, is suitable only for cases arising from portal obstruction. The great omentum is sewn into the parietal wound, and serves to form a new anastomosis between the portal and systemic veins.

MORISON'S OPERATION.—The liver is scarified and adhesions form with the under surface of the diaphragm. Usually combined with similar treatment to the spleen, i.e., Talma-Morison operation. Abdomen aspirated after operation to prevent disruption of the incision.

SUBPHRENIC ABSCESS

Definition.—A localized collection of pus in contact with the under surface of the diaphragm.

Anatomical Division.—The peritoneum is reflected from the upper surface of the liver on to the diaphragm in a cruciform manner at the coronary, falciform, and lateral ligaments. This affords a simple classification:—

1. Intraperitoneal—right anterior, right posterior, left anterior, left posterior.
2. Extraperitoneal—right, left.

Aetiology.—The greatest number occur between twenty and thirty years, the sexes being equal. The causes are septic infection from a neighbouring viscus, or very rarely traumatism.

Ruptured Gastric and Duodenal Ulcers cause about one-third or more.

Appendicitis and Hepatic Disease cause one-sixth each.

The remaining third of the cases are distributed among the following: Parturition, pyæmia, splenic infarct, thoracic disease, gastric cancer, disease of the lumbar vertebræ, cholangitis, suppurating gall-bladder, suppuration of the right kidney, stabs or crushes of the upper abdomen.

Pathology.—The process consists of either: (1) A local peritonitis, which spreads from a neighbouring viscus by contiguity or gravitation, or by the lymph-stream towards the diaphragm; (2) A cellulitis which extends beneath the peritoneum from the liver or cellular tissue of the loins.

The Bacteria are the *B. coli* and staphylococci in most cases.

Description of the Varieties (*Figs.* 151, 152, 153)—

1. RIGHT ANTERIOR INTRAPERITONEAL ABSCESS.—Over one-third of all cases. Between the right lobe of the liver and the diaphragm, in front of the right coronary and to the right of the falciform ligaments. It spreads down into the loin and below the liver, so that in the majority of cases more than one fossa is involved.

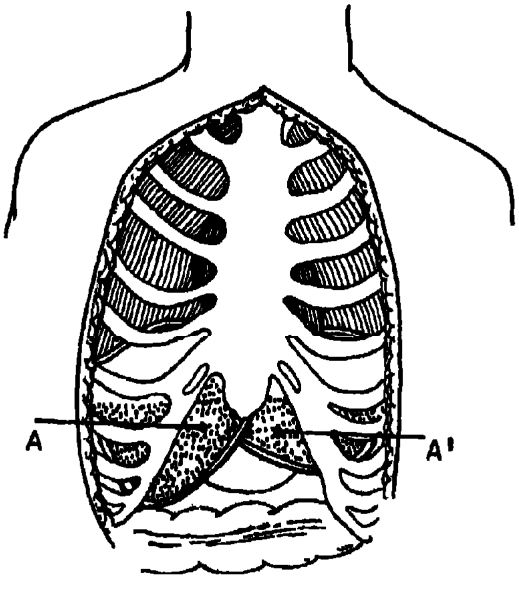


Fig. 151.—Composite diagram of a subphrenic abscess. A, Right-sided abscess; A¹, Left-sided abscess.

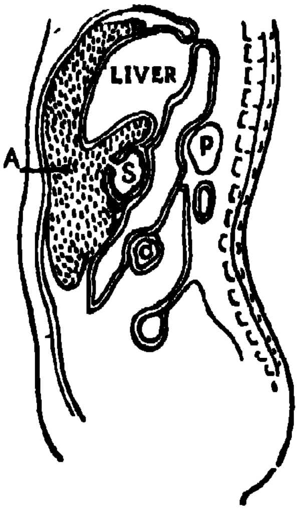


Fig. 152.—Diagram of anterior type of subphrenic abscess: median vertical section. A, Abscess; C, Colon; P, Pancreas; S, Stomach, with perforation on its anterior wall.

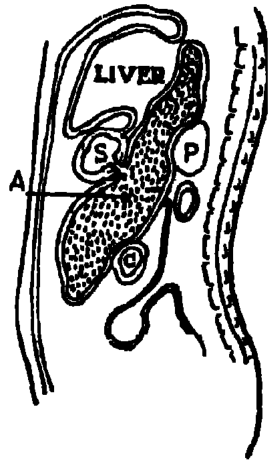


Fig. 153.—Diagram of posterior type of subphrenic abscess. A, Abscess in the lesser sac of peritoneum; C, Colon; P, Pancreas; S, Stomach, perforated on its posterior wall.

Subphrenic Abscess—Varieties, *continued.*

Causes.—Appendicitis, liver and gall-bladder suppuration, gastric and duodenal ulcers, are the commonest, and occur in this order. The liver becomes adherent to the anterior parietes, and therefore does not become displaced.

Signs.—The abscess forms between the right lobe of the liver and diaphragm, pushing the latter up and causing signs of consolidation at the base of the right lung.

2. RIGHT POSTERIOR INTRAPERITONEAL ABSCESS.—About one-eighth of the cases. Is between the right kidney and right lobe of liver behind right coronary ligament; is nearly always associated with the right anterior form of abscess

Causes.—Appendicitis in the great majority.

Signs.—An abscess extending below the right costal margin towards the right iliac fossa.

3. LEFT ANTERIOR INTRAPERITONEAL ABSCESS.—Forms about half the cases Lies between the diaphragm above, the left lobe of the liver and stomach behind, the adhesions between omentum and parietes below, the spleen to the left, and the falciform ligament to the right. Usually simple.

Causes —Gastric ulcer in the majority.

Signs.—An epigastric swelling, bounded by the mid-line and a line from the navel to the left costal margin, in which gas forms a movable area of tympanites.

4. LEFT POSTERIOR INTRAPERITONEAL ABSCESS is the rarest of all varieties (4 per cent). It is contained in the lesser peritoneal sac, having the liver above, the transverse mesocolon below, the stomach in front, and the pancreas behind.

Causes.—Posterior gastric ulcers. Disease of the pancreas and bile-duct.

Signs are very obscure, being those of a swelling behind the stomach having the relations of a pancreatic cyst.

5. RIGHT EXTRAPERITONEAL ABSCESS.—It forms between layers of right coronary ligament above back of liver, and pushes liver down towards abdomen. The space in which it forms is continuous in front with that between the layers of the falciform ligament, and behind with the retroperitoneal cellular tissue round the right kidney.

Causes.—Hepatic and biliary suppuration in the majority of cases. Suppuration of the right kidney or head of pancreas, duodenal ulcer, and thoracic suppuration rarely.

Signs.—Consolidation at the right base, with marked downward displacement of the liver. The abscess may point in the epigastrium or in the right loin.

6. LEFT EXTRAPERITONEAL ABSCESS.—A rare variety (5 per cent). It forms between the layers of the left coronary ligament.

Causes.—Suppuration of left kidney. Posterior gastric ulcer. Disease of the lumbar vertebræ Left empyema.

Signs.—Consolidation at the left base, with an abscess pointing in the left loin.

Symptoms.—

History of previous symptoms, of dyspepsia, or liver disease, is given in most cases, but the appendix cases usually are of acute origin without previous attacks.

ONSET is sudden in more than half the cases, including nearly all the intraperitoneal varieties. It is insidious in rather less than half, including all the extraperitoneal cases.

PAIN is situated over the site of the abscess, and is acute and stabbing in acute cases, dull and aching in the insidious ones.

Vomiting.

Diarrhœa follows temporary constipation.

Pyrexia, with all its constitutional signs: complexion, tongue, sweating, etc. Swinging temperature.

Rigors may occur and are of very ill omen.

Leucocytosis is constant and well marked.

Signs.—

ABDOMINAL.—An abdominal EPIGASTRIC OR HYPOCHONDRIAC SWELLING is found in two-thirds of all cases In the majority of these it is formed by an intraperitoneal abscess; in the rest by the liver pushed down by an extraperitoneal abscess The swelling does not move on respiration because it is fixed by adhesions

A TYMPANITIC AREA which moves with the patient's posture forms in front of the abscess in many cases.

TENDERNESS AND RIGIDITY are well marked over hepatic area.

THORACIC.—DULLNESS AT ONE OR BOTH LUNG BASES, with diminished breath-sounds, due to consolidation or compression of the lung, with or without pleurisy, friction sounds being heard in the former case. Pleural effusion may occur at the lung base of affected side.

THE HEART may be displaced upwards, lateral displacement being much more characteristic of pleurisy

THE ABSCESS MAY POINT either in the hypochondrium, or, more rarely, in the epigastrium or loin.

THE ABSCESS MAY BURST into: (1) The stomach, (2) Pleura; (3) Bronchus; (4) Peritoneum; (5) Intestine, (6) Externally. When this occurs into a viscus or mucous canal it often leads to cure, but into a serous cavity it is rapidly fatal.

Diagnosis is made by attention to the history and signs of abdominal disease.

IN PLEURAL EFFUSIONS, especially pyopneumothorax, the history and signs are those of lung disease. The heart is displaced laterally and not upwards. The breath-sounds are more widely abolished, and the area of amphoric breathing is much larger.

IN HEPATIC TUMOURS AND ABSCESSES the liver moves with respiration, except in malignant disease, and there is much less tenderness or hectic.

IN PNEUMOCOCCAL PERITONITIS WITH PNEUMONIA the peritoneal condition is usually generalized; otherwise it would, in fact, constitute a subphrenic abscess.

Prognosis.—About half of all cases die—all cases which are not operated on. The prospect after posterior operations is rather better than after anterior ones.

Treatment.—Free opening and drainage directly the diagnosis is made. The operations may be conducted by various routes according to the position of the abscess; only the first two routes are common.

Subphrenic Abscess—Treatment, ***continued.***

ANTERIOR ABDOMINAL ROUTE, for anterior intraperitoneal collections, the general peritoneal cavity not being opened. A supplementary drain through the loin is often required, especially in right-sided cases.

POSTERIOR ROUTE.—The incision follows the line of the 11th and 12th ribs, which are resected; the diaphragm is incised below the level of the pleural reflection, the perinephric fold is opened, and the abscess is exposed and drained. The transpleural route is not employed if possible, as mortality is unduly high.

OTHER ROUTES.—Anterior transpleural, lateral transpleural, subpleural, and lumbar openings may be required. The last is often necessary to drain collections of pus in the subhepatic pouch.

Chapter XXXV

DISEASES OF THE APPENDIX

ACUTE APPENDICITIS

Anatomy of Appendix.—

LENGTH.—Four inches, or anything between three-quarters of an inch and nine inches

DIAMETER.—Quarter of an inch (unless distended).

POSITION.—Base joins cæcum one inch below the ileocæcal valve: Point marked on surface, middle of a horizontal line between right anterior superior iliac spine and the mid-line.

It is found in the following positions, in the order of frequency named, when inflamed. (1) At the ileocæcal angle; (2) Behind the cæcum; (3) In the pelvis; (4) On the outer side of the cæcum; (5) Incarcerated in one of the retroperitoneal fossæ; (6) In front of the cæcum; (7) In the sacs of inguinal, femoral, or umbilical herniæ

DIRECTION. (1) Upwards and outwards behind the colon; (2) Downwards and inwards over the pelvic brim; (3) Upwards and inwards behind the ileum

GUIDE TO POSITION.—The muscular tæniæ coli converge to it.

STRUCTURE.—The following layers are found from within outward:—

1. Mucous membrane, containing simple mucous glands.
2. Submucous tissue, containing large solitary lymph follicles, each lymph follicle having a basal lymph sinus surrounding it on one side.
3. An ill-developed muscularis mucosæ.
4. A layer of circular muscle fibres.
5. A layer of longitudinal muscle fibres. In one or two places 4 and 5 are deficient, and through this 'hiatus muscularis' the submucous and subperitoneal layers are in direct communication, and by this channel bacterial infection often takes place.
6. Subperitoneal connective tissue containing blood- and lymph-vessels.
7. Peritoneum.

ARTERY AND VEIN.—Branches of the ileocolic which run behind the ileum—Branch from left ovarian is probably only a pathological variation.

MESENTERY.—The fold of peritoneum containing the artery in its free border—attached to the iliac mesentery. In 60 per cent of cases the mesentery stops two-thirds of the way along the appendix. This is the common site for perforation—i.e., the end of the mesentery.

ILEO-APPENDICULAR FOLD AND FOSSA.—A recurrent branch from the artery of the appendix to the ileum lifts up a peritoneal fold and bounds a fossa of this name.

Aetiology.—

SEX.—Males to females as four to one.

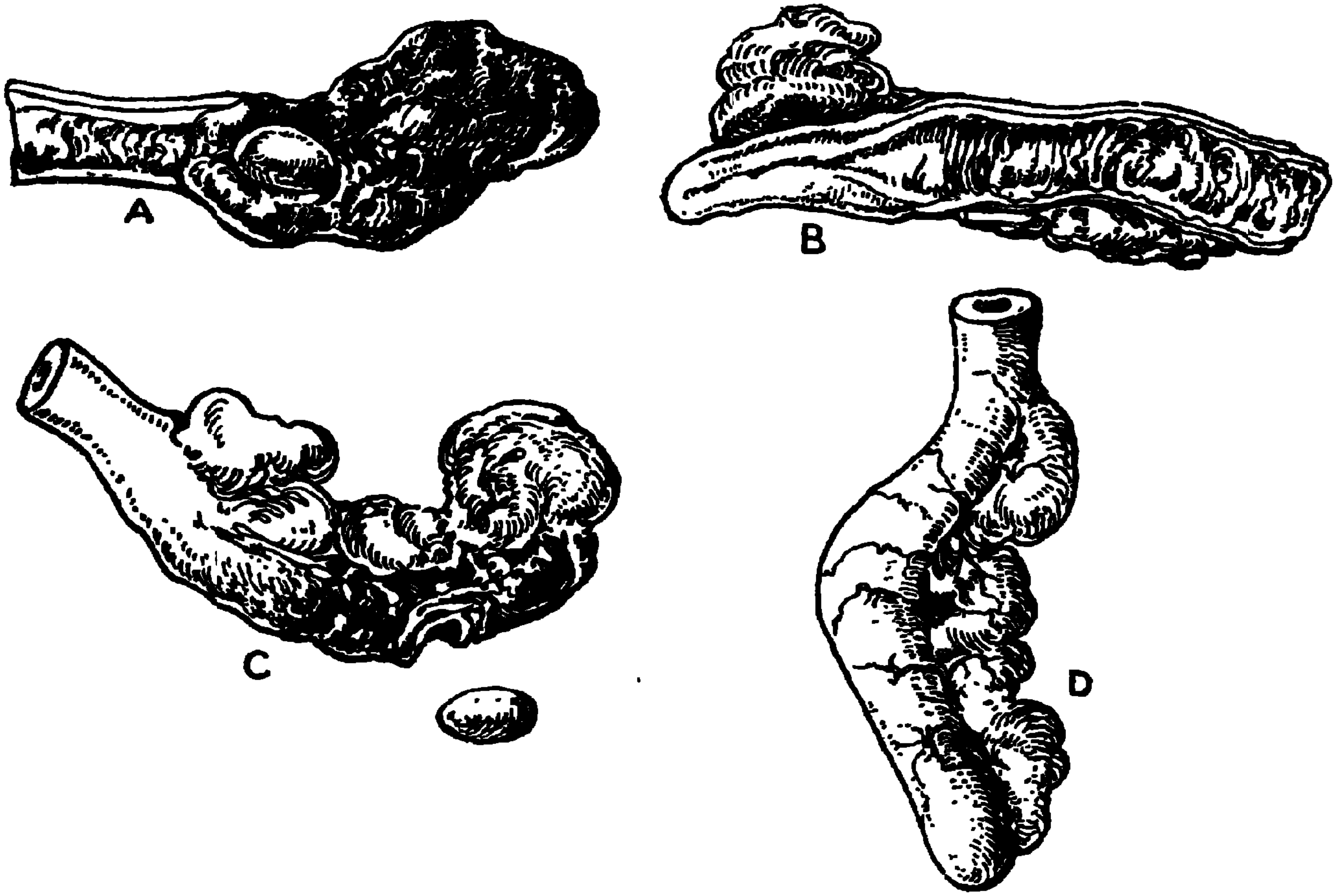


Fig. 154.—Typical conditions of appendicitis. A, Gangrene of portion beyond a concretion; B, Obliteration of tip by old inflammation; C, Ulceration and sloughing, with liberation of a concretion; D, Subacute inflammation with dilated vessels, and fat-laden mesentery.

PREDISPOSING CAUSES —

OBSTRUCTION TO THE LUMEN OF THE APPENDIX —Swelling of mucous membrane—Kinking of a long appendix—Chronic constipation—Fæcal concretions

OBSTRUCTION TO THE CIRCULATION —Owing to vessels lying behind the cæcum Owing to the artery not running out to the end of the appendix

FOREIGN BODIES —Very rare

EXCITING CAUSES —

MICRO-ORGANISMS —*Bacillus coli* and staphylococci are common Streptococci tubercle, and actinomycosis are rare

ULCERATION OR INFLAMMATION of the mucous membrane gives entry to the above

Pathological Anatomy (*Fig* 154)

EROSION of mucous membrane by concretion, foreign body, or kinking

MUCOUS MEMBRANE becomes generally inflamed or ulcerated Sloughing from pressure thrombosis, or toxic action of bacteria

BACTERIAL INVASION occurs through the bases of the mucous glands, or generally through the base of an ulcer Thence the lymphatic tissue is invaded, and by this means the process spreads to the other coats and eventually to the peritoneum Bacterial infection of the peritoneum may occur without demonstrable perforation ulceration, or gangrene

ARTERY IS OFTEN THROMBOSED This will cause gangrene, as there are no collaterals to carry on circulation

VEINS MAY BECOME THROMBOSED —Portal pyæmia and hepatic abscess may result This is exceptionally rare

PERITONEUM —Plastic or suppurative inflammation

WALL OF THE APPENDIX AS A WHOLE undergoes (1) Coagulation necrosis, or (2) Sloughing or (3) Perforation

CONCRETIONS occupy the lumen in a large proportion of the worst cases They are usually formed of a mass of bacteria agglutinated round some fæcal residue In many cases they bring about ulceration, perforation, or gangrene

FOREIGN BODIES, e g , pins, worms, or fruit seeds, are rarely present, and then may be related to the cause of the inflammation.

THE LUMEN, in addition to being occupied by concretions, may undergo one or more of the following changes —

1 It may be obliterated by destruction of the mucous membrane and the union of the submucous tissue This rarely affects the entire organ
2 Stenosis may be produced at one point, as in the last case, or by kinking
3 Dilatation of the lumen beyond the stenosis by either mucus or pus

STRUCTURES IN THE NEIGHBOURHOOD — (1) Plastic peritonitis, (2) Suppuration limited by adhesions, (3) Diffuse peritonitis

EXTENSION —The cæcum, ileum, and peritoneum in iliac fossa are almost always involved Omentum is generally involved Pelvis and uterine appendages are often involved Lower abdominal contents often. Whole of the abdominal contents rarely.

Acute Appendicitis, *continued.*

Varieties.—

1. Appendicitis without involvement of the peritoneum.
2. Appendicitis with plastic peritonitis.
3. Appendicitis with localized suppurating peritonitis.
4. Appendicitis with generalized peritonitis.
5. Relapsing appendicitis—probably a succession of attacks similar to 1, 2, or 3.

Clinical History.—

GROUP 1.—WITHOUT PERITONEAL INVOLVEMENT.—

PAIN.—Sudden, sharp, and recurring at intervals; felt in right iliac fossa. Tenderness over this region. Usual to get pain referred to umbilical region preceding pain in right iliac fossa.

TEMPERATURE.—Little or no rise.

DIGESTION.—Loss of appetite, furred tongue, constipation.

ANATOMY.—Appendix is long, or kinked, and the mucous membrane is often ulcerated and the wall invaded by bacteria.

COURSE.—Mild relapsing attacks.

GROUP 2.—WITH PLASTIC PERITONITIS.—

ONSET, in this and succeeding types.—General abdominal pain, colicky and often referred to the umbilicus or epigastrium Later, vomiting once or twice, and then after an interval local signs appear as given below.

FEVER.—Temperature rises to 101° or 102° F.

LOCALLY.—Rigidity of muscles of right iliac fossa, with a palpable mass at a later date. Most marked after third day.

TENDERNESS at first over region of ileocæcal valve, one-third distance between right anterior superior iliac spine and umbilicus—McBurney's point.

COURSE.—May subside and clear up in about three weeks. Very liable to relapse.

ANATOMY.—Local plastic peritonitis matting together cæcum, appendix, ileum, and omentum.

GROUP 3.—WITH LOCALIZED SUPPURATING PERITONITIS.—

ONSET.—Generally abrupt and severe. Often in the early morning during sleep. Sometimes preceded by malaise and indefinite pain and tenderness.

LOCALLY.—General abdominal distension and tenderness, most marked in right iliac fossa.

Right iliac fossa presents: (1) Muscular resistance and tenderness for first three to five days, (2) Indefinite resistant mass for third to seventh day; (3) Mass becomes dull to percussion.

Rectal examination shows marked tenderness on right side; boggy or fluctuating swelling later.

Right leg drawn up if posterior abdominal wall muscles, as psoas, are irritated.

TEMPERATURE.—Rises to about 104° F., with occasional rigor. Afterwards either: (*a*) Stays high, especially with gangrene; or (*b*) Drops while pulse increases.

RESPIRATION.—Almost entirely thoracic. Increased rate.

PULSE.—Rises to 100–120. Occasionally remains slow throughout.

ANATOMY.—Appendix is usually perforated or gangrenous, but suppuration may occur in the absence of gangrene, perforation, or ulceration. A collection of stinking pus is surrounded by adhesions.

ABSCESS SPREADS: Down into the pelvis; up behind the colon to right kidney; through the abdominal wall; into upper abdomen.

ABSCESS BURSTS: Into bowel, cæcum, ileum, or rectum. Very rarely it may point under the skin, or into peritoneal cavity.

DIGESTIVE ORGANS.—Tongue furred and dry. Constipation generally absolute. Vomiting severe at first and subsiding later. Small quantities of mucus, pus, or undigested food can often be detected in the motions. Rarely blood is passed by the bowel.

MICTURITION is sometimes painful, and this generally indicates that the inflammatory process is adjacent to the bladder.

COURSE.—Acute symptoms last about five days. Then usually subside and give place to those of a local abscess, or merge into (1) General peritonitis, or (2) Portal pyæmia.

GROUP 4.—WITH DIFFUSE PERITONITIS.—

ONSET and early symptoms as in last group.

LOCAL SIGNS—Except for the right lower quadrant being most tender, there are no local signs. Signs of general peritonitis rapidly set in: abdominal distension, immobility, rigidity, and tympanites.

TEMPERATURE.—High at first, generally falls, or may be subnormal during the rest of the illness.

PULSE rises to 120, and gets more rapid and smaller.

ANATOMY.—Appendix is perforated or gangrenous. Thin, stinking, seropurulent exudation lies between the coils of intestine, unlimited by any adhesions.

COURSE.—Patient dies in three to six days of general peritonitis.

GROUP 5—RELAPSING APPENDICITIS.—

RECURRENT ATTACKS of inflammation or of plastic peritonitis in or round a damaged appendix.

SYMPTOMS or signs are similar to (1) or (2).

RECURRENCE occurs at intervals of months or years. Intervals tend to become shorter. Recurrent attacks may lead to an abscess; seldom or never lead to diffuse peritonitis.

ANATOMY.—Appendix may be: (*a*) Kinked, stenosed, dilated, its walls being invaded by bacteria, the peritoneum quite unaffected; or (*b*) Inflamed and bound down in a mass of adhesions.

Diagnosis.—

GENERAL SIGNS.—Abrupt onset of generalized abdominal colicky pain, then vomiting, then, with involvement of peritoneum, appearance of local signs. Tongue is furred, and constipation is the usual thing.

LEUCOCYTOSIS with a greatly increased proportion of polymorphonuclear cells is always present with a peritoneal exudation or suppuration. In appendicitis it is never absent except in the mild cases where the peritoneum is not involved, or the fulminating cases when the infection is so intense that there is no power of vital reaction.

LOCAL SIGNS.—Tenderness, pain, and resistance in right iliac fossa are the cardinal signs of appendicitis. Tenderness and swelling felt per rectum are of great help in diagnosis in a fat or distended subject.

Acute Appendicitis—Diagnosis, *continued.*

THE DIAGNOSIS OF MILD OR RELAPSING CASES has to be made from:—

Biliary Colic.—Jaundice. Often no rise of pulse or temperature. Tenderness in upper right quadrant. Radiation of pain through to the back between the shoulders.

Renal Colic.—Pus, cells, or crystals in urine. Affection of micturition. Pain shooting into genitals or leg.

Movable Kidney.—Kidney is felt to be mobile. Symptoms referable to micturition.

Oöphoritis.—Enlarged, tender, or prolapsed ovary. Often disturbance of menstruation.

Visceroptosis with large floppy cæcum, often with Jackson's veil.—Attacks of pain in this condition are more frequent, of shorter duration, or may be constant pain in the right iliac fossa with frequent exacerbations. Relieved by recumbency.

THE DIAGNOSIS OF LOCAL SUPPURATIVE PERITONITIS from:—

Pelvic Abscess, especially parametritis. Uterus is felt fixed and displaced.

Iliac or Psoas Abscess.—Signs of bone disease. Abscess is 'cold'.

New Growth of Cæcum.—History of constipation and diarrhœa. Abdominal distension of long standing. Mass is hard, well defined, and not tender.

Early Intussusception.—Mass is not specially tender Alters in size, shape, consistency. Passage of bloody mucus per anum

Colitis —Passage of large quantity of mucus or casts per rectum. Tenderness, if present, is over the whole course of the colon.

Inflamed Uterine Appendages, e.g., pyosalpinx or small ovarian cyst.—By bimanual vaginal examination.

THE DIAGNOSIS OF ACUTE DIFFUSE PERITONITIS from perforating gastric, duodenal, or intestinal ulcer; acute pancreatitis; acute intestinal obstruction, ruptured gall-bladder; ruptured extra-uterine gestation; ruptured pyosalpinx.

Prognosis is always a matter of doubt.

BAD SIGNS —Absence of local resistant mass. Persistent high temperature. Temperature low, with rapid pulse. General abdominal distension. Persistent hiccup. Absence of leucocytosis, in the presence of grave signs of infection

Treatment.—

GENERAL.—Bed. Heat to abdomen Low diet, e g., whey or meat juices. Enemata: soap-and-water or turpentine.

DRUGS.—Opium only as a preliminary to operation. Not when diagnosis is uncertain. Purgative medicine is always dangerous, because it is liable to cause perforation of an inflamed appendix, by causing active peristalsis. This is especially the case in children and it should never be given to them.

OPERATION, with removal of appendix and suture of abdominal wall without drainage within the first 48 hours of the attack, is the treatment to be aimed at.

There is still a great deal of speculation as to the correct treatment of acute appendicitis. (1) All early cases: Immediate appendicectomy indicated (2) Acute appendicitis with spreading peritonitis without localization. Remove appendix without delay, especially in children (in whom the omentum in poorly developed) and the aged unless there is some contra-indication, e g, cardiac disease, etc (3) Acute appendicitis with localization The treatment of this group has produced much discussion One school advocates removal of appendix at whatever stage it is seen unless it is obviously resolving on its own Others suggest removal after the acute pathological processes have resolved, i e, interval appendicectomy Usually agreed that within first 48 hours removal of appendix is justified, after this, expectant school of treatment treats the case by the *Oschner-Sherren method*. Briefly this consists of (*a*) Treatment conducted in a hospital or nursing home under guidance of trained staff (*b*) Physical signs carefully charted hourly pulse-rate—this is the important indication as to progress of disease (*c*) Nil by mouth, no aperients, no morphine After fifth day, if pulse satisfactory, small fluid feeds by mouth (*d*) Heat locally to abdomen, intravenous drip saline, bowels opened by small glycerin enema if not open after fifth day If there is a rise in pulse-rate of more than 10 points during first 24 hours, or vomiting, diarrhœa, or recurrence of pain as opposed to tenderness, then delayed treatment should be abandoned and appendicectomy performed If the condition resolves without incident, appendicectomy is usually performed after an interval of 3 months

DANGER OF POSTPONEMENT OF OPERATION is especially great in children Normal temperature and relief from the primary pain are no grounds for non intervention, because both these often accompany gangrene of the appendix

PERSISTENT SINUS OR FÆCAL FISTULA AFTER OPERATION —This may be due to (1) Failure to remove the appendix, (2) Part of the appendix having been left, (3) Base of appendix remaining open into the wound, (4) Sloughing of wall of the cæcum, (5) A fæcal concretion left behind, (6) An infected non-absorbent ligature Most of these conditions can be dealt with by opening up the wound Otherwise ileo-colostomy through a median incision will be necessary

CHRONIC APPENDICITIS

Distinguish carefully between recurrent subacute appendicitis and chronic appendicitis Chronic appendicitis is relatively rare and produces reflex symptoms in stomach and duodenum—appendicular dyspepsia. Appendix shows fibrosis of the distal portion and obliteration of the lumen May be shown radiologically that the appendix does not fill adequately owing to obstruction.

Treatment.—Removal.

TUMOURS OF THE APPENDIX

True carcinoma of appendix does occur, but is very rare Carcinoid or argentiform tumours are met with, usually in young females, they are benign and do not metastasize.

CHAPTER XXXVI

INJURIES AND DISEASES OF THE STOMACH

Examination of the Stomach.—

INSPECTION AND PALPATION may reveal a tumour, or peristaltic contractions More elaborate methods of distension and auscultation have been superseded by X-ray examination.

FRACTIONAL TEST MEALS.—

Rehfuss's modification of Einhorn's tube is swallowed by patient first thing in the morning, having had no food for 12 hours previously. This tube is rubber, the size of a No. 6 catheter, having a perforated bulbous end containing a weight This is swallowed until at least 16 inches from teeth.

The whole of the fasting juice is withdrawn, the nature and amount of this is charted, and it is tested for presence of bile, starch, blood, total acidity, and free HCl.

A pint of test gruel is then swallowed with tube *in situ*, and 10 c.c. of gastric contents is withdrawn with a syringe attached to free end of tube every quarter of an hour until stomach is empty.

Each specimen is tested for blood, bile, mucus, and starch, and the amount of free HCl and total acidity estimated. The records from the analysis of these specimens is charted against the time (*Figs.* 155, 156).

In the normal stomach the acidity drops when meal is first taken, and then steadily rises owing to gastric secretion, to fall again as stomach empties and secretion diminishes. The normal stomach empties in about 2 to 2½ hours.

X-RAY EXAMINATION OF OPAQUE MEAL.—A meal containing three ounces of barium sulphate, which is opaque to the rays, is given in bread and milk or gruel. The shadow shown on the fluorescent screen by the rays is observed, and the following points noted:—

POSITION OF STOMACH.—Presence of ptosis, displacement of stomach by adhesions or tumour.

OUTLINE OF STOMACH.—Tumours may show an irregular indentation on outline. An ulcer may show as a bulge or prominence of outline when filled with barium meal. Frequently opposite the ulcer there occurs a spasmodic contraction of the circular muscle coat of the stomach, showing as a marked indentation or incisura (*see Fig.* 159, p. 405).

MOVEMENTS OF STOMACH.—Normally the stomach is empty in just over 2 hours. More rapid emptying occurs with duodenal ulcer, delayed emptying in pyloric stenosis.

GASTROSCOPIC EXAMINATION, using a flexible gastroscope, is being increasingly used.

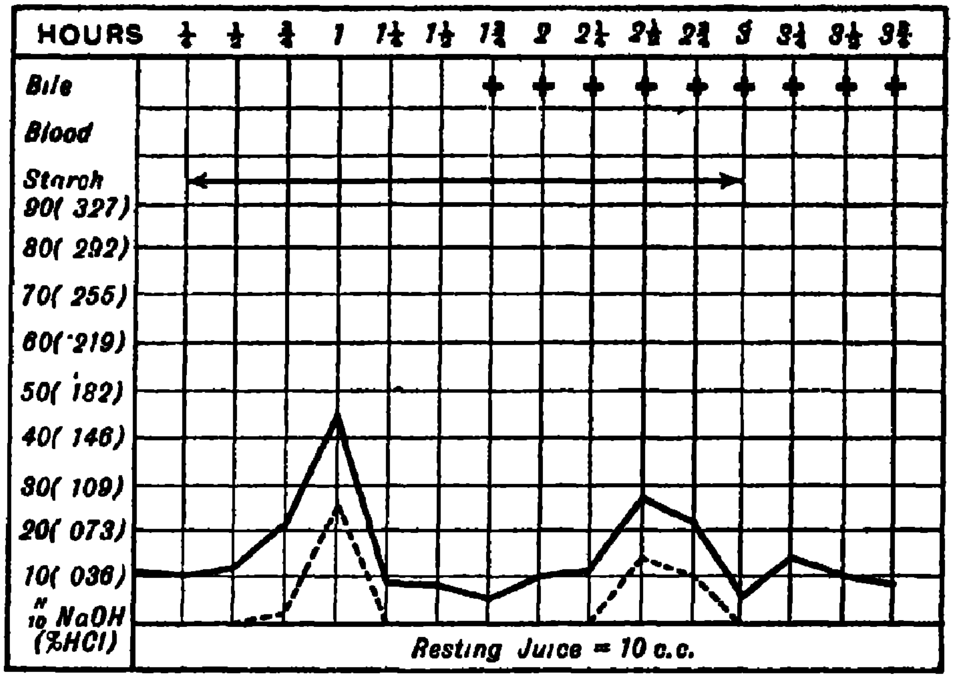


Fig 155.—Typical curve of fractional test meal in gastric ulcer.
——— Total acidity. - - - - - Free HCl.

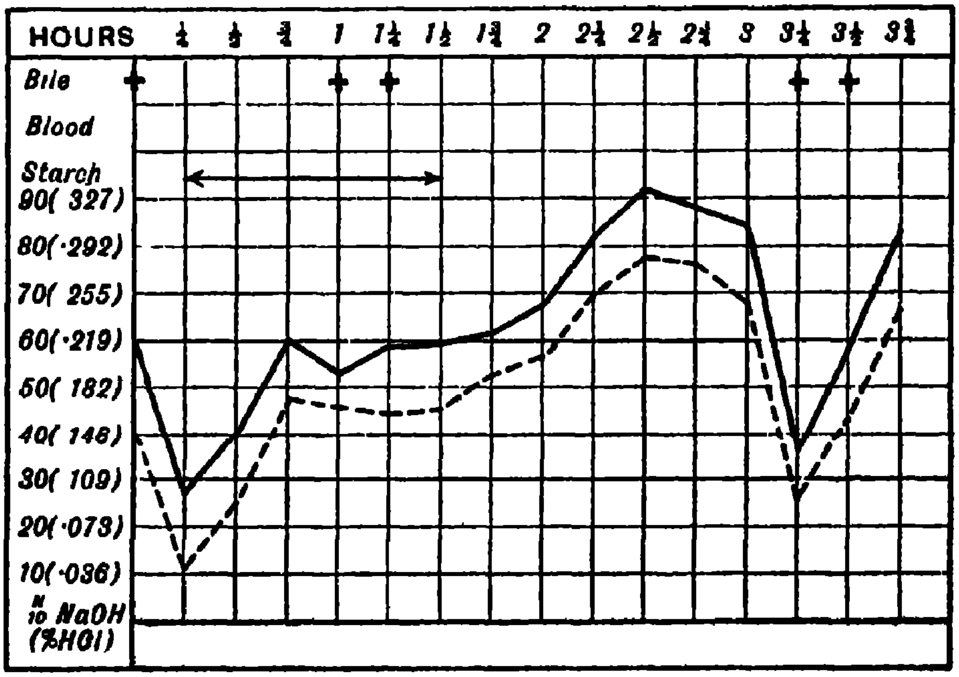


Fig. 156.—Typical curve of fractional test meal in duodenal ulcer
(*After E. F. Guy.*

FOREIGN BODIES IN THE STOMACH

Varieties.—

1. BODIES SWALLOWED BY ACCIDENT.—E.g., tooth-plates, whistles, nails by carpenters. Diagnosis usually by history aided by X rays.
2. MULTIPLE BODIES SWALLOWED BY INTENT.—Patients are usually lunatics or hysterical women.
3. BODIES THAT GROW FROM SWALLOWING MULTIPLE SMALL PARTICLES.—Pieces of hair, string, or wire swallowed at different times become welded to make a cast of the stomach. Hair ball or trichobezoar is found solely in young girls

Symptoms.—May be entirely absent, especially in small single bodies. Pain, vomiting, often streaked with blood, are common symptoms. Hair balls usually cause no symptoms until stomach is full; then pain, usually aggravated by meals, occurs; vomiting is unusual with hair ball. Occasionally a palpable tumour is present

Diagnosis.—X rays are of great value, as majority of swallowed bodies are opaque In hair ball, when opaque barium meal is given, the meal is seen around the ball, spreading out over it as if forming a cup holding something in the stomach

Prognosis.—Form of body is more important than size. Any article that can pass cardiac opening can usually pass through pylorus. Long articles like pencils, long nails, usually do not pass

Treatment.—If the article is likely to cause perforation early operation should be advised. As long as no symptoms are present, and if from the shape of the body it is thought possible for it to pass the pylorus, no treatment is required. Give mashed potatoes or thick porridge by mouth; this will tend to coat the body and aid passage through pylorus Operation of gastrotomy should not be considered before a fortnight. Gastrotomy is required if after a reasonable time body is still present, if symptoms occur, or if body is obviously unable to pass.

INJURIES OF THE STOMACH

Subcutaneous Wounds.—

CAUSES AND VARIETIES.—

1. External Trauma —Rare, direct blow in epigastrium, such as in a a run-over accident. Only occurs if stomach is full; then sudden rapid increase of pressure causes rupture to take place at weakest part of wall, i.e., along greater curve.
2. Trauma from Within.—May occur from foreign bodies within stomach, passage of œsophageal bougie; distension of stomach with gas for diagnostic purposes These causes only bring about rupture when previous disease of stomach is present.
3. Spontaneous Rupture.—Rupture of apparently normal stomach is very rare. It has occurred from severe muscular effort such as lifting or vomiting.

SYMPTOMS.—

Those of perforated gastric ulcer.

Penetrating Wounds.—

VARIETIES.—

1. Stab Wounds.—May occur in civil practice, but are very rare.
2. Shot Wounds.—Common in military practice.

SYMPTOMS AND SIGNS.—Similar to those of perforation of gastric ulcer. Hæmatemesis is common, escape of gastric juice is rare. The cause of death is most commonly severe internal hæmorrhage.

Treatment.—Early laparotomy and suture of wound of stomach.

CONGENITAL STENOSIS OF THE PYLORUS

Consists of a great hypertrophy of the muscular coat of the pylorus, with dilatation of the rest of the stomach. It is found soon after birth, usually in the first six weeks.

Pathology.—There is great hypertrophy of the muscular coat of the pylorus, almost entirely of the circular fibres. This hypertrophy is wholly concentric so that the lumen of the pylorus is greatly diminished but seen externally there is no increased diameter of the pylorus. The mucous membrane is thrown into longitudinal folds. The hypertrophy ends abruptly at the duodenum, but fades gradually away towards the body of the stomach (*Fig.* 157). The rest of the stomach is dilated and its walls somewhat hypertrophied.

Aetiology.—The cause is still uncertain. Theories advanced are:—

1. CONGENITAL ABNORMALITY —Unlikely, as usually no symptoms are present at birth. Not usually associated with other congenital abnormalities.
2. A RESULT OF SPASM —Cause of spasm said to be due to: (*a*) Incoordination of muscle fibres so that no relaxation occurs, (*b*) Hyperacidity, (*c*) Hyperadrenalism.

Clinical Features.—It is much more common in male than female children. The child at birth is usually quite healthy and of normal weight. There is usually an interval of 1 to 5 weeks or more before symptoms begin.

VOMITING —At first slight and after each meal, later becomes characteristic of pyloric obstruction—namely, vomiting of large amounts at relatively infrequent intervals, 3 to 4 feeds being returned with each vomit. It is projectile, so that the contents of the stomach may be ejected a considerable distance.

LOSS OF WEIGHT is continuous and extreme. The skin is shrivelled and dry.

CONSTIPATION is marked.

VISIBLE PERISTALSIS in an obviously distended stomach is seen, especially after giving a feed.

PYLORIC TUMOUR.—This is the most characteristic sign, and is pathognomonic of the condition. It is palpable in 80 per cent of cases. It is felt as an elongated tubular mass about 1½ inches long and ½ inch in diameter at the site of the pylorus. It is often felt best immediately after a feed, and by supporting the child on the palm of the hand back upwards.

Treatment.—Medical treatment in the past resulted in a mortality of at least 80 per cent. General trend of opinion to-day is that when the condition is diagnosed operation should be done.

Congenital Stenosis of the Pylorus—Treatment, ***continued.***

RAMMSTEDT'S OPERATION has superseded all others. This consists in the complete division of the whole length of the hypertrophied area by a longitudinal incision down to but not including the mucous membrane, allowing this to bulge out (*Fig.* 157). No sutures are used except for the abdominal wall. Careful post-operative nursing is essential.

ACUTE DILATATION OF THE STOMACH

A condition of rapid and enormous dilatation of the stomach, often ending fatally.

Aetiology.—

1. AS A POST-OPERATIVE COMPLICATION.—It occurs most commonly after abdominal operations, especially on the gall-bladder, appendix, and pelvic organs; rarely after operations on the stomach. It may follow operations on the limbs and after local anæsthesia.
2. AS A COMPLICATION OF AN ACUTE ILLNESS.—In pneumonia and typhoid.
3. FOLLOWING INGESTION OF A LARGE MEAL.—There may be no previous operation or illness.

Pathology.—Post mortem there is an enormous dilatation of the stomach, often appearing to fill the whole abdominal cavity It is U- or V-shaped, with a sharp kink at the lesser curve. The stomach wall is very thin and stretched. The dilatation does not stop at the pylorus, but involves the duodenum, often stopping abruptly at the point on the third part of the

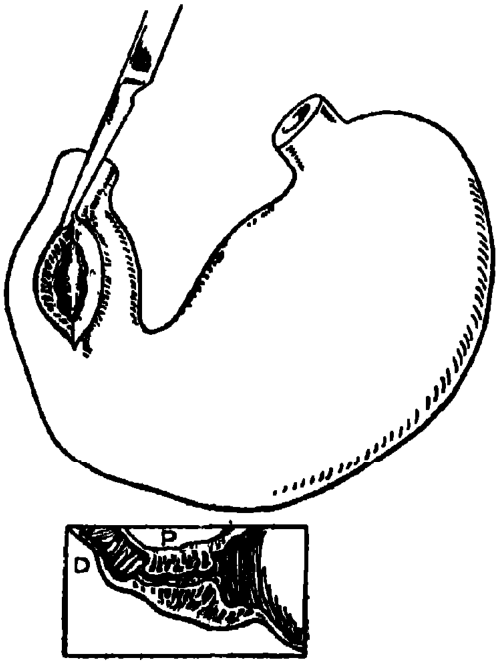


Fig. 157.—Congenital stenosis of pylorus. Lower figure shows the pylorus in section, with hypertrophy of the muscle coat. P, Pylorus; D, Duodenum. Upper figure shows Rammstedt's operation, the peritoneum and muscle being divided, whilst the mucous membrane is preserved.

duodenum where the superior mesenteric vessels cross. In other cases the dilatation fades away into the ascending portion of the third part of the duodenum approaching the duodeno-jejunal flexure.

Causation.—Various theories suggested. It may be due to :—

1. EXCESSIVE SECRETION.—This was thought to be so rapid that the stomach became paralysed. This theory does not explain the cause of the secretion, or the fact that the greater part of the distension is due to gas, not fluid.
2. SPASM OF THE PYLORUS.—This does not account for the dilatation of the duodenum.
3. OBSTRUCTION OF THE DUODENUM BY THE SUPERIOR MESENTERIC VESSELS.—This view is supported by the fact that in about a third of the cases the dilatation ceases abruptly at the point of crossing by the vessels.
4. PARALYSIS OF THE STOMACH primarily, and then pressure of the dilated stomach on the duodenum.—Box and Wallace showed that post mortem a normal stomach can be enormously dilated with water by forcing it in from the cardiac end. The water does not pass through the duodenum even if the jejunum be cut across, or even if the superior mesenteric vessels and peritoneal folds in the neighbourhood be cut through. If, however, the distended stomach be lifted off the duodenum, the contents of the stomach pass easily through the duodenum. This is now the generally accepted view. It is a paralytic condition of the stomach possibly due to pulling on the sympathetic plexus round the cœliac axis

Symptoms and Signs.—These usually do not arise until the second or third day after an operation—i e., until after the anæsthetic sickness has passed off.

PAIN AND DISCOMFORT IN EPIGASTRIUM.—Often the first sign. Is constant, and is a feeling of distension. Not usually severe.

VOMITING —This is the most marked feature. At first of small quantities at infrequent intervals, later becomes more or less continuous. Vomited material is at first undigested food, then bile-stained, and later black or brownish from blood and bile.

DISTENSION OF THE STOMACH —Distension of the epigastrium comes on early: most marked at first to left of middle line above the umbilicus; later becomes extreme. Outline of the distended stomach may be visible.

SPLASHING OF STOMACH CONTENTS.—Readily obtained. Visible peristalsis is rare.

PASSAGE OF STOMACH TUBE allows escape of large quantities of gas and fluid, often with such suddenness as to resemble an explosion. Stomach fills up again almost at once.

GENERAL CONDITION OF PATIENT rapidly deteriorates. Collapse soon comes on, with intolerable thirst.

Diagnosis.—Severe prolonged post-anæsthetic vomiting, vomiting from peritonitis, or from intestinal obstruction, is differentiated by the absence of localized abdominal distension, succussion splash, and the characteristic evacuation of large quantities of gas and fluid by the stomach tube.

Treatment.—Operations such as gastro-jejunostomy or gastrostomy are almost useless. The usual treatment is as follows. The patient is laid prone with a pillow under the chest and another under the pelvis, and the foot

Acute Dilatation of the Stomach—Treatment, ***continued.***

of the bed is raised. This position is maintained for as long and as often as the patient can stand it, e.g., 15 minutes every 2 hours. Frequent repeated evacuation of the stomach contents by stomach tube. One c.c. of pituitrin is given 8-hourly, with $\frac{1}{100}$ gr. eserine 2-hourly for six doses. It is important to give intravenous salines to replace fluids and chlorides lost. Occasionally one lavage of the stomach is sufficient to cure the condition, but often two or three attempts are necessary, and it is advisable to leave the stomach tube in situ until all signs of dilatation have passed.

ULCER OF THE STOMACH

Ulceration of the stomach occurs in the following conditions:—

1. Acute gastric ulcer or erosion.
2. Chronic gastric ulcer.
3. As a part of acute or chronic gastritis.
4. From injury by foreign bodies or chemical erosions.
5. Carcinomatous or sarcomatous ulcers.

ACUTE GASTRIC ULCER OR EROSION

Pathology.—The ulcers are usually multiple, and mostly found on the posterior surface and fundus of the stomach. They are small, 2 to 3 mm. in diameter, long or oval in shape, and have a characteristic sharp edge as if punched out of the mucous membrane The floor is smooth, and usually formed by the muscular coat; there is no infiltration. They heal readily without contraction. They tend to open the blood-vessels of the submucous coat During life no change can be seen on the peritoneal coat at the site of the ulcer, and after death they require careful search to see them, being best shown by holding up the stomach to the light and looking for light patches. These represent the ulcers

Clinical Features.—Acute gastric ulceration is found clinically in the following conditions:—

1. AS A COMPLICATION OF SOME OTHER DISEASE —Acute ulcers may occur in any septic intoxication, such as in acute suppurative appendicitis, or septicæmia, etc. Usually the history is that the patient is going downhill from some septic lesion, during the course of which, after a feeling of nausea, he vomits blood; this may be profuse, repeated, and fatal The hæmatemesis may be preceded by a few days' epigastric pain and vomiting after each meal. There may be no sign or symptom of gastric ulceration, yet post mortem multiple acute ulcers may be found.
2. AS A CAUSE OF SEVERE HÆMATEMESIS —There may be a few days' or weeks' history of gastric disturbance, such as pain after meals and vomiting, and then suddenly a large quantity of blood is vomited. This may be repeated and fatal.
3. AS A DISEASE IN YOUNG WOMEN.—Often associated with anæmia.
   PAIN in epigastrium definitely related to meals, coming on after them.
   VOMITING common, at the height of the pain. This does not give as complete a relief as in chronic ulcer
   HÆMATEMESIS very common, and may recur. Often profuse, but seldom fatal.
   TENDERNESS, both superficial and deep, common in epigastrium.

Treatment.—Surgical treatment is never required Rest in bed. Lenhartz's diet Hæmatemesis from an acute ulcer should be treated by medical means

CHRONIC GASTRIC ULCER

Pathology.—

ULCER —*Often very large, frequently several inches in diameter* *Usually single, multiple only in 20 per cent or less* *Most commonly found in pyloric part of stomach, lesser curvature, and posterior wall of stomach, in that order of frequency* (*Fig* 158)

SHAPE —Round or oval

EDGES —Rounded, indurated, and heaped up, unlike clear-cut acute ulcer Shows marked infiltration, so that whole mass may resemble carcinoma lymphatic glands never enlarged however

BASE —Formed by sclerosed tissues adherent to stomach, commonly pancreas or liver

DEPTH —Considerable so that usually the peritoneal coat is involved

PERITONEAL COAT —Shows small vascular points around edge of ulcer radiating into surrounding peritoneal surface Often on surface is a fibrinous deposit, giving rise to adhesions

The ulcer produces great contraction by healing In its extension it may open a large blood-vessel outside the stomach, e g , the gastroduodenal or splenic artery

Causation.—The reason for an acute ulcer becoming chronic is not understood Bolton found great difficulty in making an acute ulcer become chronic experimentally repeated injection of gastro-toxic sera, increasing the acidity, feeding with bacteria when an acute ulcer was present, did not make it become chronic Ulcers occur in all parts of the gastrointestinal tract bathed in gastric juice Theories —

1 EMBOLISM —Theory advanced by Virchow on the analogy of the size and shape and appearance of infarcts elsewhere Although it is

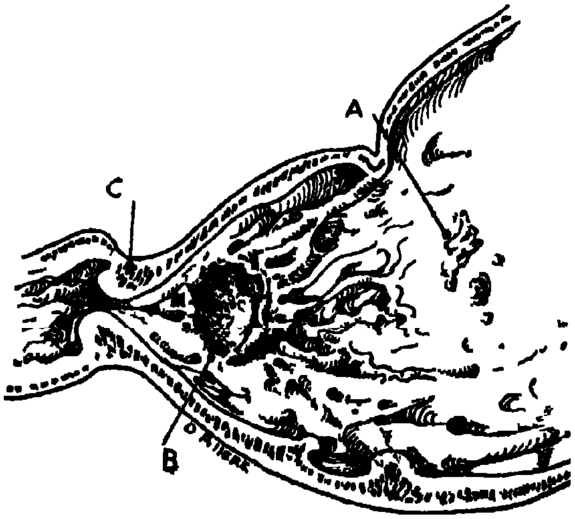


Fig 158 —Gastric ulcer Pyloric portion of stomach seen in longitudinal section A, Recent ulceration, B, Chronic ulcer in early stage of malignancy, C, Hypertrophied pylorus (× ½)

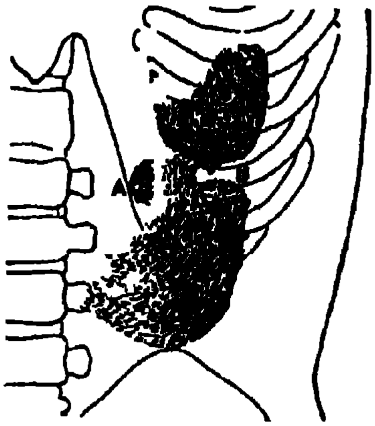


Fig 159 —Radiogram after barium meal in case of chronic gastric ulcer A, Niche representing crater of ulcer on lesser curve, B, Incisura or notch caused by spasm of stomach opposite ulcer

Chronic Gastric Ulcer—Causation, ***continued.***

possible to reproduce ulcers by artificially occluding vessels of the stomach, it is rare to find ulceration of the stomach in conditions where emboli of kidney and spleen are common, except where a definite septic element is present.

2. THROMBOSIS.—There is some evidence to show that thrombosis of gastric vessels is followed by ulceration, but usual type of ulceration is not associated with general arterial degeneration, nor does it occur in patients suffering from peripheral thrombosis.
3. NEUROPATHIC DISTURBANCE —In spinal lesions and injuries of the cœliac plexus, and after section of the vagus, ulcers may occur. They have been known to occur also in lesions of the hypothalamus possibly affecting the parasympathetic centre.
4. BACTERIAL INFECTION —There is a considerable amount of evidence to show that acute ulcers are directly dependent upon a septic infection, e.g., in the mouth, tonsils, and especially the appendix. The path of the infection is probably by way of the blood-stream. It is probable that an inflammation of the lymphoid follicles arises by infection from the original focus; these become enlarged and inflamed and later necrotic, giving rise to ulcers.
5. SPECIFIC GASTRIC POISONS —Bolton showed that by injecting the gastric cells of one animal into another of the same or different species he was able to prepare a gastro-toxic serum. If this serum was injected into an animal of the same species as that from which the gastric cells were obtained, acute ulcers rapidly formed in the stomach. These ulcers were due to the acidity of the gastric juice after the cells had been injured by the gastro-toxic serum, since if the HCl of the gastric juice was neutralized by giving doses of sodium bicarbonate before the injection of the serum, these ulcers did not form.

Aetiology.—Men suffer more often than women—83 men to 81 women (Moynihan). Age, 30 to 50.

Symptoms.—

PAIN.—

SITE.—In epigastrium or lower thorax. Later in the history it radiates to whole epigastrium, and later still to back between the shoulder-blades.

RELATION TO FOOD.—This is extremely constant; the pain appears at a definite time after each meal, varying from ½ to 2½ hours. The time is constant in each attack and after each meal. It never comes on while food is being taken or immediately after a meal, but there is always a free interval between the meal and the onset of pain

The pain continues for some time, and disappears as the stomach empties. There is always a painless interval before the next meal. The rhythm of gastric ulcer pain is: Food, comfort, pain, comfort; then food, comfort, pain, comfort. This is constant during the attack. The pain is immediately relieved by vomiting. It is also relieved by bismuth or alkalis. The site of the pain is no indication of the site of the ulcer. If pain comes on early after meals the ulcer is generally near the cardiac orifice; if late, the ulcer is near the pylorus.

VOMITING.—Only present in about 50 per cent of cases. It occurs at the height of the pain, and immediately relieves it.

HÆMATEMESIS.—Only occurs in about 25 per cent of cases.

APPETITE.—Generally good. The patient often says he would like to eat anything, but is afraid to do so on account of the pain after.

GENERAL CONDITION.—In early stages of disease patients are fat and well Later they come to look ill, and lose weight

TENDERNESS —There is usually no superficial hyperæsthesia, but deep tenderness is usually present over mid-point of pyloric plane.

The above symptoms, of which pain is the most prominent, occur in attacks. Each attack lasts 2 to 6 weeks and then disappears, whether medical treatment is given or not A period of complete relief follows for 2 to 6 months, when another attack occurs, either without cause, or after a chill or over-exertion This periodicity of attacks is extremely characteristic and almost pathognomonic of chronic ulcer. In the attack the symptoms are as described above, and the pain is constant in its appearance after each meal The history of these periodical attacks usually extends for years In the later years the attacks tend to lengthen in duration and have a shorter interval of freedom between

Signs.—

TEST MEAL —Free HCl and total acidity are slightly raised. Fractional test meal shows somewhat increased acidity and digestion lasts longer (*see Fig* 155, p 399)

X-RAY —Position of stomach is often higher than usual, being drawn up by puckering of gastrohepatic omentum.

Outline of stomach shows a crater-like niche on lesser curve (barium in ulcer cavity), opposite to which is a well-marked persistent indentation or notch on the greater curve caused by spasm (*Fig* 159) In expert hands X-ray examination gives 95 per cent correct diagnosis.

GASTROSCOPY —Exact diagnosis of size and activity and response to treatment is obtained by visualization of the ulcer by gastroscopy No ulcer may be considered healed because symptoms have disappeared, as temporary remissions are characteristic of the condition.

Treatment.—In younger subjects with a short history, medical treatment should always be given a thorough trial Healing of the ulcer must be secured as confirmed by radiology and/or gastroscopy

In older subjects with a long history of ulceration surgical measures are advised, because. (1) Healing of the ulcer is not now likely; (2) Relapse later is almost certain, (3) Complications are likely to occur. Surgical treatment may be as follows —

Gastrojejunostomy for ulcers with stenosis at the pylorus

Conservative Excision of the Ulcer for small ulcers on the lesser curve

Partial Gastrectomy is the ideal for all types, but carries a greater risk.

Combination of Excision and Gastro-enterostomy.

The Complications of Chronic Gastric Ulcer

1. Acute Complications

1. **Perforation.**—Perforation may be *acute*, leading to diffuse peritonitis and possibly later subphrenic abscess; or *subacute*, causing local peritonitis and subphrenic abscess.

Complications of Gastric Ulcer—Perforation, *continued*.

ACUTE PERFORATION.—

Frequency.—Occurs in 15 to 20 per cent of cases.

Site of Ulcer.—The anterior wall is perforated in more than two-thirds of the cases. Acute ulcers which perforate are near the cardiac end more often than the pyloric. The reverse holds with chronic ulcers.

Sex and Age.—Are those of chronic ulcer, viz., men more than women.

Size of the Perforation.—Varies from a minute aperture to one admitting two fingers.

History of Preceding Dyspepsia is usually present, but is absent in about 10 per cent of the cases.

Symptoms and Signs.—

Pain, sudden and excruciating.

Shock and collapse, rapid, severe, and increasing

Vomiting often occurs once and is rarely repeated.

Rigidity of upper abdomen: very hard and tender, and board-like.

Liver dullness is obscured by gas.

Shifting dullness occurs in the flanks.

Signs of local peritonitis occur in the subacute cases: (1) In epigastrium, especially round pylorus; (2) In region of right iliac fossa; (3) As a subphrenic abscess.

Diagnosis.—The sudden onset of acute epigastric pain with marked shock, followed by board-like and excessively tender epigastrium, in the majority of cases renders the diagnosis easy.

Treatment.—Open the abdomen in the midline above the navel. Examine the anterior gastric surface, beginning at the cardiac end, and then the posterior surface through an opening in the gastrocolic omentum. Close the perforation with a double layer of stitches. If this is impossible, sew omentum over it. Perform gastro-jejunostomy only if pyloric stenosis renders this imperative, and this is exceedingly rare, 0·5 per cent of cases.

Prognosis after Operation depends on: (1) Condition of stomach—the fuller the stomach the worse the outlook; (2) Time between operation and perforation—if less than twelve hours the outlook is good, if more than thirty-six it is almost hopeless in acute cases; (3) Size of the perforation.

2. Hæmorrhage.—

CAUSES (of hæmatemesis in general).—

Trauma.—Blow or foreign body.

Gastric Ulcer.—Acute or chronic.

Other Surgical Conditions.—Cancer, aneurysm, appendicitis.

General Diseases.—Hepatic cirrhosis, purpura, scurvy, cardiac disease, leukæmia, enteric fever, septicæmic conditions.

HÆMORRHAGE FROM ULCERATION is, apart from traumatism, the only form amenable to direct treatment.

Source of the Bleeding is from capillaries in most acute ulcers, and from arteries in the chronic ulcers and the most severe and fatal forms of bleeding. The splenic, gastro-duodenal, and right gastro-epiploic arteries are those most often eroded.

In Acute Ulcer only the small vessels are eroded, and there is no induration to prevent their natural closure.

Treatment.—(1) Absolute rest. Sucking ice. Ice to the epigastrium. Feeding entirely by the rectum for some days. Morphine for restlessness. Adrenaline chloride, 1–1000 solution, 10 min. by mouth. Blood transfusion. Later, Lenhartz diet. (2) Operation is rarely necessary, and only when the above has failed. The stomach must be opened and the bleeding area ligatured. A gastro-enterostomy should be performed also, to keep the organ at rest.

IN CHRONIC ULCER there are usually signs and history of a long-standing dyspepsia. The bleeding may be insidious at first, like that of secondary hæmorrhage. When severe it comes from eroded arteries which cannot contract or retract because they are buried in adhesions.

Treatment.—Medical treatment as above whilst hæmorrhage continues; when stopped, and patient's general condition is better, then operate.

2. Chronic Complications

1. Adhesion to the Liver and Pancreas.—This and the succeeding three complications give rise to a characteristic change in the symptoms of chronic ulcer, in that the symptoms lose their periodicity. For years there may have been a regular periodicity in the occurrence of each attack, but when the ulcer begins to erode the liver or pancreas, the attack lasts much longer, six months or more. In addition, other features are:—

PAIN is more continuous throughout the day, but still increased by food. It is much more severe, and now often radiates more widely, even up to the left shoulder

VOMITING as before, and when present still gives complete relief to the pain.

GENERAL CONDITION is distinctly poorer. Patients often show loss of weight and anæmia, thus suggesting carcinoma.

X-RAY EXAMINATION is often characteristic, revealing the pocket of the ulcer fixed in liver or pancreas.

DIAGNOSIS.—Often difficult from carcinoma.

TREATMENT.—Gastrojejunostomy has not had such success with this complication as direct attack on the ulcer, whether by excision, cautery, or knife, with gastrojejunostomy. Partial gastrectomy gives the best results. Gastrojejunostomy in 'Y' is suitable for large adherent ulcers incapable of treatment by other methods. Temporary improvement can be obtained by jejunostomy in the very wasted case. Later, more radical measures may be undertaken.

2. Pyloric Obstruction.—

CAUSES.—

1. Gastric and duodenal ulcer.
2. Gastric tumour.
3. Perigastric inflammation with adhesions.
4. Carcinoma of stomach.

CLINICAL FEATURES.—When occurring as a complication of chronic gastric ulcer there is usually a long history of periodic attacks of gastric ulcer symptoms in which, as the ulcer is near the pylorus, the onset of pain after meals occurs late. The attack lasts longer and longer with the onset of obstruction until the characteristic symptoms appear.

PAIN is much less, but constant throughout the day. It is still increased by food.

VOMITING is now constant in the attack, and occurs at infrequent intervals (once in a day or two days); it is large in amount, the contents of several meals being vomited.

Complications of Gastric Ulcer—Pyloric Obstruction, ***continued.***

GENERAL CONDITION becomes poorer.

PHYSICAL SIGNS of distension of the stomach may be seen, with visible peristalsis. Splashing is easily elicited. A tumour of the thickened pylorus may be felt. X rays show great gastric retention.

TREATMENT.—Pyloric stenosis usually means that the ulcer is healed and the symptoms are purely due to the cicatrization. Gastro-enterostomy therefore gives the most satisfactory results. If signs of active ulceration are still present or any suspicion of malignant change, then gastrectomy should be performed.

3. Hour-glass Constriction.—

CAUSES.—Perigastric adhesions resulting from an ulcer. Perforation and adhesion of an anterior ulcer. Cicatrization of a transversely placed median ulcer. Carcinoma, either primary, or secondary to an ulcer.

ANATOMY.—The stomach is divided by a transverse constriction into two equal or unequal parts The constriction is usually about the middle; it may be so narrow that a catheter can scarcely pass its lumen. Great puckering and scarring from ulceration are present, and indicate the causation.

CLINICAL FEATURES.—

SYMPTOMS.—Loss of periodicity of simple ulcer. Pain more constant and of increased severity. Vomiting now more frequent If cardiac pouch small, regurgitation takes place after a few mouthfuls, resembling œsophageal obstruction If cardiac pouch large, due to constriction being nearer pylorus, vomiting resembles that of pyloric obstruction. Almost all cases due to chronic ulcer occur in women; those occurring in men are all due to carcinoma.

SPECIAL PHYSICAL SIGNS —

1. Fluid introduced through a tube cannot be all returned.
2. A gush of putrid fluid after washing the stomach clean.
3. Splashing felt after the stomach has been emptied by siphonage.
4. X rays show two cavities separated by a narrow channel of considerable length. This is the only reliable investigation, as it gives valuable information as to the size of the pouches present.

TREATMENT.—

1. GASTROPLASTY.—A division in the long axis of the stomach of the stricture, which is sewn up transversely. Only suitable in the absence of adhesions and induration.
2. GASTRO-ANASTOMOSIS.—Making a large anastomotic opening between the two pouches. It is successful only in the absence of pyloric stenosis, and only if the ulcer has healed.
3. GASTRO-ENTEROSTOMY.—Uniting the cardiac compartment to the jejunum.
4. PARTIAL GASTRECTOMY.—Necessary in malignant and advisable in some simple cases.

4. **Onset of Carcinoma.**—The frequency with which carcinoma occurs in a chronic ulcer is variously estimated. Figures varying from 4 to 80 per cent are given as the proportion of all cases of carcinoma of the stomach in which evidence of previous chronic ulcer exists; probably the true incidence is nearer the lower figure. When occurring as a complication of

ulcer, there is a loss of periodicity of the symptoms, the last attack lasting some months with more or less constant pain, though still increased by food. The pain may not be so severe, but signs of general deterioration of the patient soon occur.

DUODENAL ULCER

Duodenal ulceration is similar to that of the stomach, and often occurs at the same time, but there are certain important characteristic differences.

FREQUENCY is about a quarter, or less, that of gastric ulcer

SITUATION.—Almost always in the first part of the duodenum; remainder may occur as far down as the level of ampulla of Vater.

NUMBER.—Often multiple, one being opposite to another.

GASTRIC CONDITION.—In about 5 per cent there is an associated gastric ulcer.

AGE.—All ages are liable.

SEX.—Males are much more frequently affected than females: about four to one.

Symptoms are usually similar to those of gastric ulcer, pain, vomiting, and excess of free HCl being the rule (*see Fig.* 156, p. 399). Special characteristics are:—

1. Many cases are quite latent and only discovered post mortem
2. Many cases are quite latent until a severe perforation or bleeding occurs.
3. THE PAIN is dull and aching or burning, felt to the right of the midline. It occurs three to four hours after meals, and often appears to be relieved by food ('hunger pain'). Attacks of pain lasting for two or three weeks are followed by intervals of freedom from pain lasting for months. The attacks are more frequent in cold weather. The pain comes on with great regularity at the same time every day.
4. BLEEDING is not so frequently observed as in gastric ulcer, because it is not looked for in the fæces. It may occur as hæmatemesis or melæna. Or it may be quite masked in severe or fatal cases, the patient bleeding to death into his own intestine. The gastroduodenal or right gastro-epiploic arteries are those most often eroded

Varieties.—As in gastric ulcer, they are either ACUTE, with a special liability to perforation, or CHRONIC, with liability to hæmorrhage and cicatrization

Complications.—

HÆMORRHAGE.—Is more likely to be fatal, and less amenable to medical treatment, than in gastric ulcer.

PERFORATION.—It is more frequent and more fatal than in gastric cases. It is very liable to be mistaken for acute appendicitis, because the exudation gravitates into the right iliac fossa.

In subacute or chronic cases a subphrenic or other ABSCESS, or an internal or external FISTULA, may occur.

CICATRICIAL CONTRACTION produces symptoms and signs indistinguishable from those of pyloric obstruction, and the condition must be treated in the same way.

OCCLUSION OF BILE-DUCTS, with lithiasis and jaundice. } Very rare.
PANCREATITIS, from occlusion of the pancreatic duct. }

CARCINOMA is so rare a development of duodenal ulcer as to be practically negligible.

Duodenal Ulcer—Complications, ***continued.***

Treatment.—In younger subjects medical treatment must be given a thorough trial. Healing of the ulcer demonstrated by radiology must be secured—relief of symptoms is insufficient. When medical measures fail, then surgical intervention is necessary. If a young subject, the ulcer is clearly vicious in type and radical measures—partial duodenectomy, partial gastrectomy, etc.—are necessary. Gastro-enterostomy in older subjects with low acidity.

SIMPLE TUMOURS OF THE STOMACH

Varieties.—Adenoma. Myoma. Cysts. These are all very rare, but may cause signs of: (1) A very movable epigastric tumour; (2) Attacks of vomiting caused by the tumour catching in the pyloric aperture.

ADENOMA occurs as a polypoid mass with a narrow pedicle. Possibly it is a precursor of malignant disease.

MYOMA may grow inside or outside the viscus.

CYSTS grow between the coats of the stomach or between the layers of omentum. They are probably formed by a budding off of an outgrowth from the stomach.

TREATMENT.—Removal, with careful microscopical examination.

SARCOMA OF THE STOMACH

These form about 5 per cent of all malignant gastric growths

Varieties.—Lymphosarcoma or spindle-celled.

SYMPTOMS AND SIGNS are the same as in carcinoma, but gastric dilatation is not so common. Also it is more usual for a large movable tumour to be formed than in the case of cancer The spindle-celled variety may form a polypoid mass capable of local removal.

CARCINOMA OF THE STOMACH

Aetiology.—More common in men than women. Age: forty to seventy includes most of the cases; may occur at thirty, or even younger. Chronic gastric ulcer is sometimes its precursor.

Anatomy.—

REGION AFFECTED —Pylorus in 60 per cent; lesser curve, and cardiac end, about 10 per cent each; other sites together, 15 per cent; general, 5 per cent.

NAKED-EYE APPEARANCE —

1. COMMON TYPE —Annular growth round the pylorus Pyloric canal obstructed or obliterated. Growth ends abruptly at the duodenum; extends farther along lesser than greater curve. Stomach wall is thickened to half an inch or more. Muscle is hypertrophied in and near the growth. Mucous membrane is often destroyed by ulceration. Peritoneal coat is thick and puckered.
2. SOFT FUNGATING friable mass, with infiltrated base.
3. EXCAVATED ULCER, with everted edges.
4. GENERAL INFILTRATION and contraction of the whole organ—the 'leather-bottle stomach'.

HISTOLOGY.—

1. SPHEROIDAL-CELLED CARCINOMA.—Over 60 per cent. Generally of a scirrhous type. Columns of spheroidal cells invade all coats.
2. COLUMNAR-CELLED CARCINOMA.—Rather less than 40 per cent. Bases of the gastric glands invade the deep tissues. Growth consists of atypical glandular alveoli.

   Both these forms are liable to certain modifications: (*a*) Colloid degeneration; (*b*) Encephaloid; or (*c*) Scirrhous type.
3. SQUAMOUS-CELLED CARCINOMA growing from the œsophagus.

EXTENSION.—

To THE LYMPH-GLANDS along the lesser curve; in the portal fissure of liver; round cœliac axis; and finally to left supraclavicular glands.

To THE PERITONEAL SURFACE, and thence by adhesions to liver, pancreas, colon, peritoneal cavity, anterior abdominal wall

THE GREAT OMENTUM early becomes permeated with cancer cells.

THE PELVIC CAVITY may be engrafted by secondary growths comparatively early (probably by trans-cœlomic implantation), and Douglas's pouch should always be examined for such growths.

Masses of glands may obstruct the portal vein and thoracic duct.

SUBMUCOUS TISSUE is always invaded much more than the muscular or serous coats, and it is in this tissue that the most advanced edge of the growth is found.

LESSER CURVE.—The most marked extension in most cases is along the lesser curve, following the line of the lymph-stream which leaves the stomach with the coronary artery (*Fig* 160).

DUODENUM.—Usually, even in advanced cases, the growth stops abruptly at the pylorus.

Symptoms.—

1. CARCINOMA OF LESSER CURVATURE.—

a. INSIDIOUS ONSET.—First stage is often not referred to stomach, but patient first complains of weakness and loss of energy, loss of weight

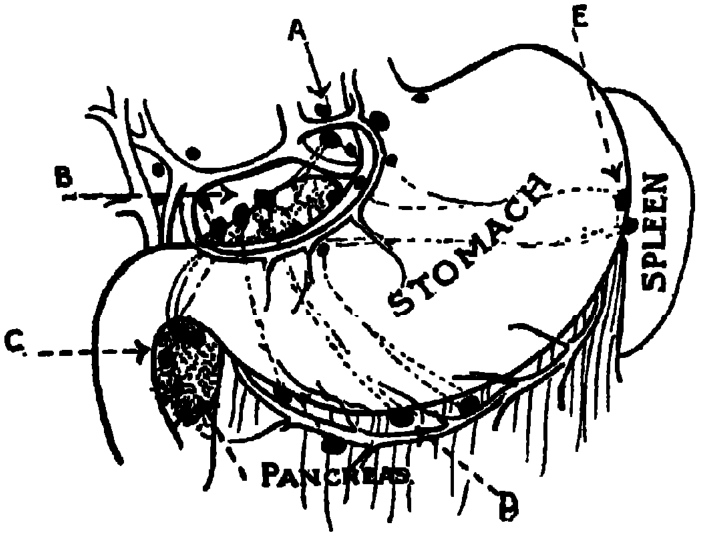


Fig. 160.—Lymphatics of the stomach. A, Glands round coronary artery; B, Glands along lesser curve; C, Subpyloric group; D, Group along greater curve; E, Group at the fundus. Note that A, B, and C are the important groups involved in the spread of cancer of the stomach. All of these are liable to involve the pancreas by adhesion and invasion.

Carcinoma of the Stomach—Symptoms, *continued*.

and appetite. Later there appears some discomfort or sense of fullness of ill-defined nature after meals. This becomes more or less constant, but is accentuated after meals. Later, vomiting or regurgitation of small quantities of foul fluid occurs, although this may be entirely absent. The symptoms progress until emaciation, presence of gastric tumour, or secondaries make the diagnosis clear. Jaundice from secondary deposits (in the portal fissure) may be the outstanding feature.

b. FOLLOWING GASTRIC ULCER.—The previous history is that of many years of periodical attacks of typical chronic gastric ulcer. With the onset of carcinoma there is a loss of periodicity, the last attack being different from the others: it has lasted longer, and the pain is now constant throughout the day, though still being made worse by food. The pain as a whole is not so severe as previously. The symptoms outlined under (*a*) above appear.

2. CARCINOMA OBSTRUCTING THE PYLORUS—There will be a history of a few month' epigastric pain, more or less constant, but increased by each meal and worse at the end of each day. Vomiting is early and is large in amount, shows evidence of decomposition, and occurs at relatively long intervals. It gives some relief to the pain. There is loss of appetite and marked loss of weight. On examination, visible peristalsis and splashing will be present, and a tumour is able to be palpated The symptoms are thus those of a chronic ulcer obstructing the pylorus, but of more rapid progress, and with a history of only a few months' preceding dyspepsia, instead of, as in chronic ulcer, many years of dyspepsia.

3. CARCINOMA OBSTRUCTING THE CARDIAC ORIFICE.—The condition starts insidiously: loss of energy, loss of weight, then pain in epigastrium occur. The pain is present after each meal, and may persist throughout the day. Vomiting occurs early, and consists of a regurgitation of stomach contents. Later, symptoms of œsophageal obstruction arise, the pain occus during the actual swallowing of food, and there is difficulty in the passage of food.

4. SILENT GROUP.—Patients seek advice for general malaise, tiredness, and anæmia. Nothing to point to a carcinoma of stomach. Discovered during routine examination.

Physical Signs.—

TUMOUR.—Small, movable, and at level of 9th costal cartilage to the right of the midline in a pyloric growth. Indefinite epigastric mass indicates involvement of the body of the stomach, probably lesser curve. The mass, when seen early, moves easily up and down on respiration and less so from side to side.

GASTRIC CONTENTS.—Absence or diminished free HCl is found in about 80 per cent of cases. HCl is usually present in cases developing on site of a chronic ulcer. There may be presence of lactic and butyric acids, sarcinæ. Boas-Oppler bacilli, and particles of growth. The vomit rarely contains free blood, but often contains intimately mixed altered blood of brown colour known as ' coffee-grounds vomit'.

X-RAY EXAMINATION.—This shows impaired motility of stomach wall, with a filling defect at site of growth. This is a valuable means of diagnosis.

IN LATE STAGE.—There is excessive emaciation, lemon or jaundiced skin, and loss of elasticity of skin. In the abdomen there is a hard mass in the epigastrium, with enlargement of the liver. Ascites. Œdema of legs. Severe pain in back and epigastrium. There may be a mass of glands in the left supraclavicular region from secondaries traversing the thoracic duct.

Diagnosis.—Ought to be made in the early stages if possible. In the majority of cases, when the second stage is well marked radical treatment is impossible. At the present time exact diagnosis is made by: (1) Barium meal examination; (2) Gastroscopy (valuable in early doubtful cases); (3) Laparotomy. In cases of doubt laparotomy should be always advised.

IN SIMPLE CHRONIC DYSPEPSIA, the wasting is not marked. There is no definite alteration in gastric contents.

IN SIMPLE DILATATION of the stomach or with an ulcer, HCl is present. Symptoms and emaciation rapidly improve with lavage.

IN INFLAMMATORY ADHESIONS round the gall-bladder, an indefinite mass may be present. A history of gall-stones or jaundice. Not much wasting. HCl present in stomach.

DOUBTFUL CASES—All cases in patients over forty-five in whom pain and wasting do not yield to treatment, should be submitted to an exploratory operation.

Treatment.—

PALLIATIVE.—(1) Drugs. (2) Diet: (3) Where obstruction is marked—gastro-enterostomy.

RADICAL.—Partial gastrectomy. This should be undertaken wherever feasible, as it offers the only chance of success. Contra-indications are: (1) Secondaries in the liver; (2) Secondaries in the portal glands; (3) Secondaries in the peritoneum; (4) Excessive fixity. When doubt exists the patient should be subjected to partial gastrectomy.

CHAPTER XXXVII

INTESTINAL OBSTRUCTION

Causes.—

USUALLY ACUTE.—(1) Strangulation by bands and through apertures, including herniæ, internal and external, (2) Kinking; (3) Volvulus; (4) Intussusception; (5) Obstruction due to foreign bodies; (6) Paralytic ileus

USUALLY CHRONIC.—(6) Stricture; (7) Tumours growing from the bowel wall; (8) Pressure of tumours outside the bowel; (9) Fæcal accumulation.

Sex and Age —The male sex is specially prone to hernia, volvulus; the female sex to pressure by tumours, obstruction by gall-stones, and fæcal accumulation; children to intussusception and congenital stricture; patients over fifty to malignant growths.

General Pathology of Obstruction without Strangulation as produced by any form of stricture or new growth.—

THE BOWEL BELOW THE OBSTRUCTION is empty and contracted The ballooned rectum below a rectal stricture is the only exception to this.

THE BOWEL ABOVE THE OBSTRUCTION is dilated and hypertrophied in proportion to the chronicity of the case. The mucous membrane is thick, catarrhal, and ulcerated The ulcers (when the obstruction is in the colon) are specially common just above the obstruction, at the cæcum, and in the lower part of the ileum.

THE ULCERS usually cause death by perforative peritonitis, but they may form chronic fæcal abscesses or fistulæ, or very rarely a bimucous fistula, which relieves the obstruction.

SACCULATION OR POUCHING may be marked in either gut, but is commonest in the large The pouches are sometimes filled with fruit-stones. Gangrene of the gut may rarely occur above the obstruction.

General Pathology of Strangulation as produced by a hernia or volvulus.—

THE INTENSITY OF THE CHANGES and of the symptoms depends upon: (1) The site of strangulation, being much more severe in the small than in the large intestine, (2) The length of gut involved; (3) The tightness of the strangulation, i e., the degree of vascular occlusion.

THE GUT ABOVE THE STRANGULATION is red, congested, and distended with gas and fluid. Ulcers or gangrene above the site of strangulation are very rare.

THE GUT BELOW THE STRANGULATION is pale and empty, but very rarely may be the seat of some enteritis.

THE STRANGULATED LOOP becomes congested, œdematous, greatly distended with gas. Later its surface loses its lustre, is covered with sticky exudation, the colour becomes purple and black, and the gut gangrenous.

AT THE LINE OF STRANGULATION ulceration and linear gangrene are common, especially in the proximal loop (for further details, *see* STRANGULATED HERNIA, p. 445).

THE CHIEF FACTOR IN STRANGULATION is the vascular occlusion. This produces thrombosis of the vessels, great meteorism from decomposition of the intestinal contents, death of the tissues, with passage through them of bacteria.

Clinical Varieties.—(1) Acute; (2) Chronic; and (3) Chronic ending in acute.

ACUTE OBSTRUCTION

Causes.—Internal strangulation, kinking, volvulus, foreign bodies, intussusception, strangulated external hernia.

Symptoms.—

SHOCK.—Due to an implication of visceral nerves. General prostration; anxious, drawn face; great pallor. Subnormal temperature, cold sweats; small, soft, rapid pulse; shallow, quick respiration. Vomiting at the time of seizure

This abdominal shock is common to all forms of 'peritonism'. and at first there are few or no distinguishing features.

ITS DEGREE is in this case proportioned to. (1) The suddenness; (2) The tightness of strangulation; (3) The nature of gut strangled, small bowel giving more shock than large, and jejunum more than ileum; (4) The amount of gut; (5) The youth of the patient. It passes off or becomes less within twenty-four hours in all except the most acute cases. It is much diminished by morphia

PAIN.—Of sudden and severe onset. It has four different causes, which act in succession: (1) The actual nipping of the gut, (2) Abnormal peristaltic movements above the obstruction; (3) Distension of the gut; (4) Peritonitis

IT IS CONSTANT, BUT SUBJECT TO EXACERBATIONS in complete obstruction; it is intermittent in partial obstruction.

IT IS REFERRED TO THE REGION OF THE NAVEL, and gives no indication of the seat of the lesion.

THERE IS A CONSPICUOUS ABSENCE OF TENDERNESS at first; in fact, pressure often relieves the pain, and the patient rolls about in restless agony.

LOCAL TENDERNESS MAY APPEAR about the third day over the site of the obstruction, and quickly gives place to the general tenderness of peritonitis.

THE PAIN DIMINISHES OR CHANGES ITS CHARACTER when perforation, gangrene, septic intoxication, or peritonitis sets in.

THE PAIN IS INCREASED after aperients, food, enemata, or palpation.

VOMITING.—There are three causes of vomiting: (1) Shock causes sudden vomiting at the onset, especially when the stomach is full; (2) Obstruction of the gut; (3) Peritonitis and distension. The vomiting of obstruction is first bilious and later stercoraceous, depending on site and age of obstruction. The tongue is dry and foul

STERCORACEOUS VOMIT is due to decomposition and bacterial products. It is most conspicuous in small-gut obstruction, and occurs early in proportion to the nearness of the obstruction to the stomach. It is caused by the return flow of an axial stream of fluid from the site of

Acute Obstruction—Symptoms, *continued.*

obstruction. Its occurrence is delayed by opium. It should be looked upon as a sign of impending death rather than a sign of intestinal obstruction.

TRUE FÆCAL VOMITING is excessively rare, and is due to a fistulous communication between the stomach and large bowel.

CONSTIPATION is absolute: neither fæces nor flatus pass in most cases. Due to reflex nerve paralysis. Lower bowel may empty itself spontaneously or by enemata, but even this is unusual.

SPURIOUS DIARRHŒA, with mucus and blood, which is so frequent in chronic cases, is only seen in acute intussception among the acute cases.

TYMPANITIC DISTENSION due to meteorism is in proportion to the lowness of the seat of obstruction. When the small bowel only is involved, the central part of the abdomen is most distended, but when the colon is affected, the flanks swell out. Sigmoid volvulus gives the most extreme distension. It is not conspicuous in very acute cases (other than volvulus), but it greatly increases with the onset of peritonitis.

THE ABDOMEN remains flaccid until the onset of peritonitis. Visible peristalsis never occurs in a case which is primarily acute. Its occurrence proves a chronic cause.

TUMOUR is rarely felt in acute cases: (1) Intussusception, (2) Foreign body, (3) Matted intestine, (4) Internal hernia, may give rise to one, but except in the first they are rare.

URINE is diminished or even suppressed by shock. Later the small quantity is due to excessive vomiting. Indicanuria is usual in acute cases when the small gut is involved.

TEMPERATURE is usually subnormal throughout. A slight rise may accompany peritonitis, and an abrupt fall often follows perforation.

PULSE-RATE is at first slow, and there is a fall in blood-pressure. A rapid pulse is a grave sign.

PERITONITIS supervenes in a large proportion of fatal cases. It is caused by (1) a general transudation of bacteria through the gut, or by (2) perforation Its onset is marked by the abdomen becoming tender and rigid, the pulse hard and thready, and where perforation has occurred, gas and free fluid may give the usual signs.

TOXÆMIA may occur with or without peritonitis, and is the last stage of all cases except the ultra-acute ones who die in the stage of shock. Delirium and unconsciousness, dry and cracked tongue, sunken eyes, ashen and livid complexion, restlessness, with profuse cold sweats, uncountable pulse, sighing rspiration.

CHRONIC OBSTRUCTION

Causes.—A stenosis or obstruction of the gut, which may be due to: (1) Kinking; (2) Adhesions; (3) Some kinds of volvulus; (4) Neoplasms outside the gut; (5) Foreign bodies; (6) Strictures, innocent or malignant —this cause being commoner than all the others put together; (7) Fæcal accumulation.

Symptoms.—

ONSET is gradual, with indefinite 'dyspepsia' and ever-increasing constipation.

PAIN is colicky and paroxysmal. Tends to be localized over the seat of obstruction. It is increased by aperients.

VOMITING is irregular and inconspicuous, often absent.

BOWELS.—Constipation is the rule, but it is very important to remember that regular daily actions may occur in spite of marked chronic obstruction. Spurious diarrhœa is common in stricture of the colon or rectum. It is caused by catarrh above the stricture. The motions are then very foul. Bloody mucus is common only when the disease is low down and of a cancerous nature.

SHAPE OF THE MOTIONS.—Occasionally in sigmoid or rectal disease the motions are compressed or tape-like.

ABDOMEN.—Distension, both by gas and fæces. It is round and barrel-like, or wide and flat. Rumbling and gurgling are heard and felt.

VISIBLE AND PALPABLE PERISTALSIS is produced by. (1) Increased peristaltic efforts above a stricture; and (2) Hypertrophy of the gut. It is therefore well developed in proportion to the chronicity of the case.

CONSTITUTIONAL.—There is much wasting. The complexion becomes muddy and yellow, with yellow conjunctivæ. The temperature rises occasionally, especially after aperients. The constitutional symptoms are proportional to the chronicity of the case rather than to the nature of the obstruction.

Differences in Chronic Obstruction in the Small and Large Bowels.—

IN STENOSIS OF THE SMALL GUT.—General symptoms of indigestion. The pain is influenced by the ingestion of food and the nature of the diet. Meteorism is late. Aperients often relieve Vomiting is frequent. Tumour is felt in 30 per cent of cases of cancerous stricture. Live three to six months after the onset of symptoms

IN STENOSIS OF COLON.—Indigestion is absent Nature of food makes little difference to the pain. Aperients aggravate the symptoms, and may cause collapse, vomiting, perforation, or death. Vomiting is rare until obstruction becomes absolute, and then rarely becomes stercoraceous until the fourteenth day or later. Blood and mucus are common with spurious diarrhœa, especially in cancerous stricture. Ballooning of the rectum and tenesmus may be present when the lesion is low down. Tumour is felt in 40 per cent of cases of cancer. Live three to nine months after onset, but may live two years after colostomy.

CHRONIC OBSTRUCTION BECOMING ACUTE

Causes.—May occur in any variety of chronic obstruction, when it may be be brought on by. (1) Occlusion of a stricture by solid fæces; (2) Kinking of the gut; (3) Peritonitis at and above the stricture; (4) Purgatives; (5) Morphia.

Differences from a Primary Acute Attack.—The shock, pain, and suddenness of onset are much less. Visible peristalsis only found if a chronic obstruction has preceded the acute.

DIFFERENTIAL DIAGNOSIS

1. ACUTE ABDOMINAL DISEASES OF OTHER KINDS.—Biliary colic—Renal colic—Lead colic—Ruptured gastric ulcer—Ruptured gut —Perforated appendix—Acute pancreatitis—Twisted ovarian cyst—Extra-uterine gestation—Ruptured pyosalpinx—Torsion of the testis. The diagnosis is made clear generally after the lapse of a few hours by the development of some special symptoms.

Intestinal Obstruction—Differential Diagnosis, ***continued.***

2. ACUTE PERITONITIS from a perforated appendix, stomach, or gall-bladder. In this a rigor commonly occurs with the onset. An early rise of temperature is usual though not invariable. Tenderness is extreme, local at first, more diffused afterwards. The pain in peritonitis tends to subside sooner than in obstruction. The vomiting is less copious, less persistent, and less likely to be stercoraceous. Constipation is not so absolute. The abdomen is hard and rigid. Meteorism is early and diffuse. The patient lies still, with knees up, as compared with the restless movements of one with obstruction. The pulse is very hard, of high tension. Leucocytosis appears in the majority of cases.
3. TUBERCULOUS PERITONITIS is especially likely to be mistaken for intussusception or for chronic obstruction. There are usually some fever and local tenderness.
4. CHOLERA.—Cases of acute obstruction with profound shock following diarrhœa, or cases of acute intussusception, have been mistaken for this.
5. DYSENTERY AND MEMBRANOUS COLITIS have been confused with intussusception.
6. THROMBOSIS OF THE MESENTERIC VESSELS.—An acute abdominal seizure occurs, with pain, vomiting, and collapse. Blood-stained diarrhœa may occur. The mesenteric veins or one of the arteries may be affected. In the latter case infarction of the gut supplied arises. The diagnosis is seldom made before operation.

TREATMENT

1. PRE-OPERATIVE.—

MORPHINE.—Relieves the pain and shock. Lessens peristaltic contractions, and therefore allays the vomiting. It causes a freer flow of urine by diminishing the nerve inhibition. It prevents the rapid development of an intussusception.

Dangers.—It obscures diagnosis and tends to paralyse the bowel. It should, if possible, be withheld until the diagnosis is clear, and then given in doses of ¼ gr. in adults, and ⅛ gr. in children.

ENEMATA.—A simple soap enema, one pint in bulk, given as early as possible. Generally it is returned unchanged. It should not be repeated. If it washes out of the lower bowel it makes operative measures easier; it also aids in subsequent feeding by rectum.

PURGATIVES should in no circumstances be given if actual obstruction exists or is suspected. They will increase pain, shock, vomiting, and strangulation.

FEEDING.—All feeding by mouth is absolutely stopped. Thirst is best allayed by half a pint of water at blood heat given per rectum every four hours. A long drink of water (preferably hot) may be allowed occasionally. It causes vomiting and thus washes out the stomach.

WASHING OUT THE STOMACH often gives temporary relief from vomiting. It is specially indicated before the administration of an anæsthetic. Passage of Miller-Abbott tube to relieve the distension.

2. OPERATIVE:—

EVERY CASE SHOULD BE SUBMITTED TO OPERATION when complete obstruction is proved or probable. The prospects of success depend largely upon the EARLY PERIOD at which it is undertaken.

THE ANÆSTHETIC.—The stomach should first be washed out, to minimize the danger of inhalation of vomited matter. As little anæsthetic as possible is given. Often local anæsthesia or SPINAL ANÆSTHESIA will be desirable.

THE INCISION should be median below the umbilicus. The only cases in which other incisions are justifiable are: (*a*) When the cause is known to be cancer below the sigmoid flexure, when an incision as for inguinal colostomy will be made; (*b*) When there is a possibility of the case being one of acute appendicitis.

IN DESPERATE CASES —A mere enterostomy of the nearest available coil of distended gut is sometimes all that can be done. It may relieve the urgent symptoms and allow a more radical operation later.

SEARCH FOR THE SEAT OF OBSTRUCTION —The cæcum is first examined. If it is distended, the large bowel is at fault; if collapsed, then the small gut is the seat of obstruction. The pelvis and left iliac fossa are next examined The large bowel can easily be palpated in its whole course. The small bowel usually has to be brought out on to the abdomen.

PUNCTURE OF THE GUT.—When great distension exists and affects the colon, and especially the sigmoid flexure, a trocar is thrust in and the gas allowed to escape. A single stitch serves to close this. It is of little use to puncture distended small bowel, because this relieves only the coil punctured

ENTEROSTOMY should always be performed after the cause of obstruction has been removed, if great distension, especially of the small bowel, exists. Tie in a Paul's tube.

THE TREATMENT OF GANGRENE OF THE GUT —In desperate cases it is possible only to bring the dead parts outside and perform an enterostomy above the disease But if the patient's condition allows it, an immediate enterectomy is to be performed.

SPECIAL VARIETIES

I. INTERNAL STRANGULATION

Internal strangulation, under bands and through apertures.

Causes.—The strangulating agents are of six kinds :—

1. PERITONEAL BANDS.—These result from any form of local peritonitis, especially appendicitis, also from tuberculous disease in the intestine or glands. They become stretched and rolled into cords by intestinal movements. They are often attached to the region of the cæcum, the mesentery, the uterus and ovaries, or the umbilicus. They act in two ways: (*a*) As a short band forming an arch, beneath which the gut becomes snared; and (*b*) A long band which forms loops in which the gut is knotted.
2. CORDS FORMED BY OMENTAL ADHESIONS.—Similar to above, a strand of omentum becoming attached to cæcal region, pelvis, or hernial orifice. Commoner on left side.
3. MECKEL'S DIVERTICULUM is the remains of the vitello-intestinal duct. It is present in about 2 per cent of bodies. It is about 3 in. to 6 in. long, attached to the ileum 2 ft. to 3 ft. from the cæcum. It may have a lumen of the same size as the gut, or its lumen is obliterated, and it forms a vitelline ligament. Its end may be free, or attached to the umbilicus,

Internal Strangulation—Causes, ***continued.***

mesentery, or any other spot. It causes obstruction: (*a*) As a short band under which the gut is caught; (*b*) As a long cord which snares the gut; (*c*) As a free diverticulum with a knobbed end which knots itself round a loop of gut; (*d*) By producing kinking of the gut over it or by its traction; (*e*) By producing an intussusception or volvulus; (*f*) By a stricture formed at its junction with the gut

4. FALSE DIVERTICULA are hernial protrusions of the mucous membrane through the other walls of the gut They occur: (*a*) Towards the mesentery of the small intestine; (*b*) Into the appendices epiploicæ of the colon. The latter may perforate or become inflamed, and so give rise to obstructing bands.
5. ADHESIONS OF NORMAL STRUCTURES.—(*a*) The appendix may be adherent by its tip to the mesentery, ileum, cæcum, bladder, or parietes; (*b*) The Fallopian tube; (*c*) A part of the mesentery may form a tight band by the fixation of a portion of the gut; (*d*) Appendices epiploicæ; (*e*) Ovarian pedicle.
6. SLITS AND APERTURES.—These may occur: (*a*) In the mesentery; as the result of traumatism or congenital defect; (*b*) In the omentum, (*c*) In a membranous adhesion

The Mechanism of Strangulation.—

It is nearly always the small gut which is caught, and generally the lower part of the ileum. When once caught, (*a*) the congestion produced by pressure on the veins, (*b*) gaseous distension, and (*c*) a twisting of the loop to form a volvulus, bring about strangulation

Clinical Characters.—

AETIOLOGY —Usually young adults, because these are the patients so liable to the different forms of peritonitis, hernia, or injury. In over 60 per cent there is a history of one or other of these conditions previously.

ONSET is sudden and abrupt in the majority of cases, and sometimes is determined by a straining movement, large meal, or purgative.

PAIN is early, severe, and continuous. Vomiting is constant, copious, and severe, and later becomes stercoraceous.

COLLAPSE, with profound prostration, is well marked.

ABDOMEN shows nothing characteristic. Very rarely a local dullness or an indefinite mass is caused by the engorged coils above the obstruction.

Course of the case ends fatally in about one week, the extremes being eight hours and twenty days. Patients seldom live more than three days after onset of stercoraceous vomiting.

Prognosis.—Apart from operation it is always fatal. Death if early in proportion to the tightness of strangulation, the height of the bowel involved, the length of bowel caught.

Death is due to septic absorption, collapse, peritonitis, or perforation, in this order.

Special Points in Operative Treatment.—Bands and adhesions must be carefully divided between ligatures; diverticula removed, and the stumps sutured.

Internal Herniæ

Diaphragmatic Hernia.—In about half the cases the orifice in the diaphragm is congenital, and in the other half it is traumatic. The stomach, colon, and small intestine are involved, in this order. The symptoms may be acute or chronic. Diagnosis is practically impossible until the abdomen is opened.

TREATMENT.—This will generally involve opening the pleural cavity to close the rent.

Duodenojejunal Fossæ.—There are at least nine different varieties of these fossæ, the commonest formed by a peritoneal fold passing from the terminal part of the duodenum towards the left, and making a pouch looking upwards or downwards. The others are, chiefly, the paraduodenal, formed by the fold of peritoneum lifted by the inferior mesenteric vein, and those running under the root of the mesentery or into the transverse colon.

THE LEFT DUODENO-JEJUNAL HERNIA is eight times as common as the other. It is probably into the paraduodenal fossa It extends to the left behind the descending colon, and has the inferior mesenteric vein and a branch of the left colic artery running in front of its neck (*Fig* 161).

THE RIGHT DUODENO-JEJUNAL HERNIA extends behind the peritoneum towards the right behind the ascending colon It enters a fossa in the root of the mesentery, and has the superior mesenteric vessels in front of its neck (*Fig* 162).

THE SYMPTOMS vary, from those of dyspepsia and uneasiness, to chronic or acute intestinal obstruction. In the latter case the course is acute.

TREATMENT.—Difficult because of the large vessels in front of the neck of the sac.

Foramen of Winslow.—Very rare form, and only possible when an abnormal mesocolon exists It usually involves the large intestine, but sometimes a large part of the small gut may be involved. The gut may further break its way through the gastro-hepatic omentum or through the transverse

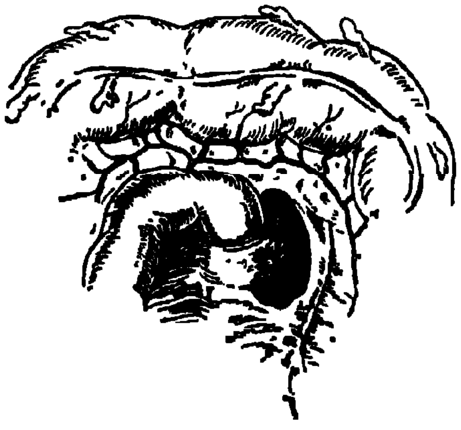


Fig. 161.—Paraduodenal, or left duodenojejunal, fossa, with inferior mesenteric vein in neck of sac.

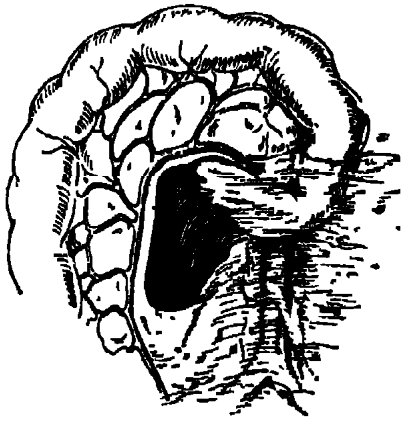


Fig. 162. — Mesentericoparietal, or right duodenojejunal, fossa, with superior mesenteric artery in neck of sac.

Internal Hernia into Foramen of Winslow, ***continued.***

mesocolon, and then become strangulated. Over the neck of the sac at the foramen of Winslow run the portal vein, hepatic artery, and bile-duct. The symptoms are usually those of chronic obstruction with epigastric distension, followed by acute obstruction.

TREATMENT.—Difficult because the neck of the sac cannot be divided.

Intersigmoid Hernia.—The intersigmoid fossa is in the root of the sigmoid mesocolon, looks downward and to the left, and is bounded by the sigmoid artery. The symptoms are those of internal strangulation.

Pericæcal Hernia.—In the cæcal region fossæ are: (1) Superior ileocæcal fossa between meso-appendix and general mesentery; (2) Inferior ileocæcal fossa between bloodless fold of Treves and mesentery of appendix; (3) Retrocæcal fossa

II. KINKING

Causes.—

A loop of bowel may be KINKED OVER A BAND. Adhesions or volvulus of the loop usually complicate this rare condition.

A loop of bowel may be KINKED BY THE TRACTION of a band or diverticulum.

ADHESIONS OF THE GUT to form V- or N-shaped loops. This is usually an after-effect of an inflamed or strangulated hernia, the symptoms coming on weeks or months after the hernia

THE CONTRACTION OF ADHESIONS may compress the gut. This occurs round the colon, especially at the hepatic flexure, from gall-bladder inflammation.

THE MATTING TOGETHER of several coils of intestine may affect the small or large bowel In the former it results from local peritonitis, and produces subacute obstruction; in the latter it results from chronic constipation and ulceration, and produces chronic obstruction

Clinical Signs.—These cases are so rare that they do not form a definite clinical group They may conform to any type of obstruction.

III. VOLVULUS

Definition.—A kinking of a loop of gut, by either: (1) Rotating round its own mesenteric axis (*Fig* 163)); or (2) Falling across the pedicle of another loop of gut

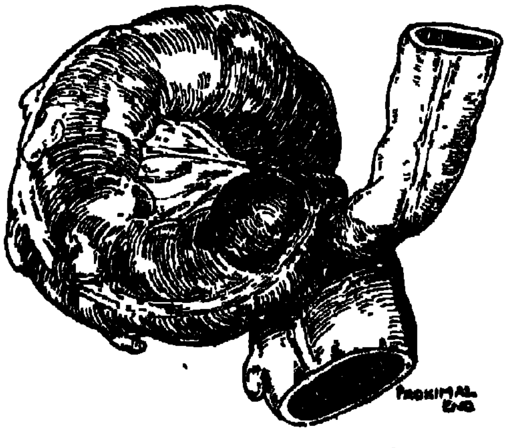


Fig. 163.—Volvulus of the pelvic colon.

Locality.—Three regions may be affected, in the following order of frequency: (1) The sigmoid loop, i.e., the pelvic colon; (2) The small intestine; (3) The cæcum and ascending colon.

The Sigmoid Volvulus.—The loop and its mesentery are usually of abnormal length; the mesenteric attachment is narrow. The peritoneum of the mesocolon is dense, contracted, and often adherent. Chronic constipation, with distension of the loop, is the prominent predisposing cause. The proximal limb usually falls in front of the distal limb. The twist may be from half to three complete turns. As long as distension exists the volvulus cannot be reduced, or re-forms after reduction. In fatal cases the sigmoid becomes enormously distended, until it presses on the liver and diaphragm, the latter being raised up to the level of the 3rd or 4th rib. The loop is intensely congested and hæmorrhagic. Its outer coats are often ruptured. Gangrene is frequent. The mesocolon is engorged. Peritonitis is of constant occurrence. The gut above is distended and sometimes perforated. Sometimes the sigmoid loop is intertwined with a loop of small intestine. The patients in the common variety are usually elderly males.

The Enteric Volvulus.—A loop of small gut has an abnormally long mesentery, or one whose attached border has been puckered by adhesion, e.g., by a caseating gland. A gall-stone in the bowel may bring about the twist. The coil, which may be from one to five feet in length, is enormously swollen, congested, or gangrenous. It is often filled with blood, but death occurs before perforation. Rarely two coils may be intertwined. Enteric volvulus occurs in patients under forty, and sometimes in children.

Cæcal and Colic Volvulus.—The rarest variety. The gut may be twisted: (1) Upon its own axis; (2) At right angles to its own axis, i.e., kinked; (3) Twisted as a loop round its mesocolic axis. The cæcum and ascending colon are the parts affected, and they are always the subjects of congenital malposition or possessed of a mesocolon. Rarely an ascending colic volvulus may be intertwined with a loop of small bowel.

Clinical Aspect of the Sigmoid Volvulus.—

AETIOLOGY.—Males are four times as commonly affected as females; usually the age is between forty and sixty. There is always a history of chronic constipation.

PAIN is early and severe, but is often intermittent or subject to exacerbations.

VOMITING is reflex in nature and is not striking—true obstructive vomiting is unusual.

ABDOMINAL CONDITION.—Rapid and enormous meteorism occurs from the distension of the sigmoid loop, which may be seen through the parietes and extends up to the diaphragm. Tenderness and rigidity are present.

RESPIRATION is embarrassed by the rapid abdominal distension.

The other forms of volvulus are too rare to permit of a clinical grouping. They usually cause acute, but sometimes intermittent or chronic obstruction.

Course and Prognosis.—Rapid (about one week) and fatal if untreated.

Death is due to asphyxia from pressure on the diaphragm, septic absorption, or peritonitis.

Special Points in Operative Treatment.—In sigmoid and colic volvulus the loop must be brought to the surface evacuated through a small incision, untwisted, stitched to the parietes to prevent re-formation of the twist, and a colostomy completed.

IV. INTUSSUSCEPTION

Definition.—A prolapse of one part of the gut into an immediately adjoining part. It causes one-third of all cases of intestinal obstruction.

THREE LAYERS OF BOWEL are concentrically placed one inside the other. Peritoneum is in contact with peritoneum and mucous membrane with mucous membrane.

The outer layer is the SHEATH or INTUSSUSCIPIENS.

The middle layer is the RETURNING LAYER, and has its coats reversed, i.e., the mucous membrane is outside towards the sheath, and its peritoneum inwards towards the inner layer.

The inner layer is the ENTERING LAYER.

The inner and middle layers, whose apposed peritoneal coats usually adhere together, are known collectively as the INTUSSUSCEPTUM.

THE NECK is the junction of the sheath and returning layer where these grasp the inner layer.

THE APEX is the junction of the middle and inner layers.

PRIMARY.—This is the common variety in infants, where no definite cause can be established

SECONDARY —Most intussusceptions in adults are secondary to a growth in the bowel wall—lipomata in the small intestine and carcinomata in the large bowel.

Varieties.—

1. ENTERIC.—Includes 8 per cent of all cases. Composed entirely of small intestine. Usually only about 6 in. in length. The jejunum is four times as liable as the ileum.
2. COLIC or RECTAL.—Includes 8 per cent of all cases Composed entirely of large intestine. Most common in the region of the sigmoid flexure. Usually quite short.
3. ILEOCÆCAL.—Includes 46 per cent of all cases. The ileocæcal valve travels down the colon, dragging with it both ileum and cæcum (*Fig.* 164). It is of large size, the apex formed by the ileocæcal valve often reaching the anus.
4. ILEOCOLIC —Includes 38 per cent of all cases The ileum prolapses through the ileocæcal valve, which together with the cæcum, remains in its place. Secondary ileocæcal invagination usually occurs later.

Anomalous or Rare Forms.—

1. Intussusception of the dying. Usually in the small gut, of small size, often multiple, may be retrograde, probably occurs during rigor mortis.
2. Retrograde intussusception is very rare, apart from those of the dying. Usually of short length, and in the colon.
3. Double, or even triple intussusceptions, are rare, and are caused by one intussusception becoming bodily prolapsed into the gut below, once, or even twice

The Mode of Growth is the same in all varieties except the ileocolic.

GROWTH TAKES PLACE AT THE EXPENSE OF THE SHEATH, i.e., more and more of the sheath becomes drawn in.

THE APEX REMAINS THE SAME THROUGHOUT, e.g., in the common ileocæcal variety, whether small or large, it is formed by the ileocæcal valve. This is due to the fact that the inner and middle layers, whose junction forms the apex, have their peritoneal coats in apposition, and these

adhere rather than glide over one another. But the outer and middle coats have mucous surfaces next to each other, and these readily glide, so that the intussusceptum slips down inside the intussuscipiens.

Pathological Anatomy.—

THE MESENTERY becomes dragged into the intussusceptum, between the inner and middle layers. (1) It is stretched by the traction of the growing intussusception. (2) It is constricted at the neck, where its bulk is largest (3) Its tissues swell by exudation of inflammatory products.

THE BOWEL ABOVE is congested and dilated, only rarely in chronic cases is it ulcerated.

THE SHEATH OR INTUSSUSCIPIENS is little changed in acute cases; in chronic cases it is affected by local peritonitis, or it may be ulcerated or gangrenous, especially opposite the apex of the intussusceptum.

THE INTUSSUSCEPTUM —

1. It is curved by the traction of the mesentery, so that its apex is directed against the wall of the sheath.
2. Œdema is most marked at the apex, which forms a globular tumour, and at the convex border.
3. Catarrh and desquamation of the mucous membrane occur, and account for the mucus and shreds in the stools.
4. Both its layers become engorged with blood, which exudes into the lumen
5. Gangrene is common in acute cases, and is most marked at the neck; in chronic cases it is most marked at the apex
6. Separation and passage of the intussusceptum may affect the whole mass, which is passed as a complete double tube a few inches to a yard or more in length, or the tube may have unfolded itself, and so present only a single layer; or it may merely come away piecemeal, the latter being more common in chronic cases

OBSTRUCTION is caused by (1) The orifice at the apex being dragged upon by the mesentery, so as to be slit-like and apposed to the sheath; (2) The curved kinking of the intussusceptum by the mesentery; (3) The narrowing of the lumen by the pressure of œdema, hæmorrhage, and inflammatory exudation, (4) The blocking of the narrow lumen by some food débris

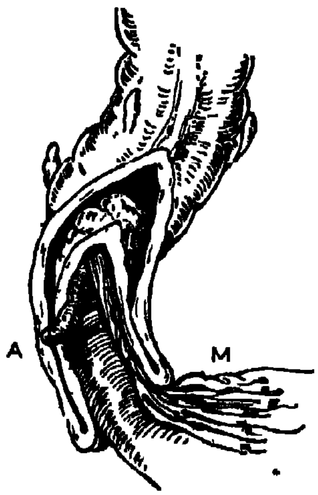


Fig. 164—Intussusception, of the common ileocæcal variety Part of the sheath and returning layer are cut away to show the entering layer. Apex formed by ileocæcal valve. A, Appendix; M, Mesentery.

Intussusception—Pathological Anatomy, ***continued.***

STRANGULATION is caused by a compression of the vessels in the mesentery, by the tissues at the neck, by swelling of the layers of the intussusceptum, and by traction and torsion.

IRREDUCIBILITY is caused by: (1) Adhesions between the inner and middle layers, these occurring as a rule in chronic cases and exceptionally in acute cases; (2) Swelling of the intussusceptum, especially at its apex; (3) Bending or twisting of the intussusceptum; (4) A polyp or growth at the apex.

Aetiology.—

SEX AND AGE.—Preponderance of males, especially in early life and in chronic cases. In acute cases 50 per cent occur before two years and 25 per cent before one year. In chronic cases 50 per cent occur between fifty and sixty.

PREDISPOSING CAUSES.—Diarrhœa, worms, masses of undigested food, polypus, new growth in the gut wall, Meckel's diverticulum, invaginated appendix (the last three very rare).

EXCITING CAUSES.—The irregular peristaltic contraction of the intestine induced by any of the above, either spasm or (rarely) paralysis causing a piece of gut to be swallowed, as it were, by the gut below

INFLUENCE OF THE ILEOCÆCAL VALVE —This acts like the sphincter ani, and tenesmus draws the cæcum over the ileum.

CAUSE OF GROWTH OF AN INTUSSUSCEPTION —When once the condition is begun the peristaltic contractions of the sheath tend to drive down the intussusceptum

Clinical Varieties.—These are: (1) Acute, which end fatally within one week; (2) Chronic, which live more than a month, seen in adults over 60 years of age.

Symptoms of Acute Intussusception.—

ONSET is sudden, with pain or tenesmus

PAIN is early. Severe at first, and may be less later when the intussusception becomes fixed. It is characteristically intermittent, the paroxysms coming on regularly and lasting a definite time. When a tumour appears the pain is localized to that region. On the whole the pain is less acute than in any other form of acute obstruction

VOMITING is frequent at the onset, but irregular and inconstant during the course of the disease It affords more relief than in other cases of obstruction. It varies inversely with the diarrhœa. It rarely becomes stercoraceous. It is most marked in enteric forms.

BOWELS.—Marked constipation is exceptional, and comes on after diarrhœa.

DIARRHŒA arises early from catarrh and excessive peristalsis; it also occurs late, and is specially offensive when the intussusceptum is sloughing.

BLOOD AND MUCUS occur conspicuously in 80 per cent of the acute cases, especially in children.

TENESMUS is often very prominent It is an early symptom, and occurs in proportion to the proximity of the lesion to the anus. Patulence of the anal sphincter may occur.

SHOCK, THIRST, DIMINUTION OF URINE, are less marked than in other forms of acute obstruction.

RIGIDITY of the abdomen and METEORISM are slight and come on late.

TENDERNESS over a localized area may indicate the position of the lesion.

ABDOMINAL TUMOUR occurs in over 50 per cent of all cases. It is most frequent in ileocæcal and colic varieties. More distinct in children. It is sausage-shaped and curved. Most often seen over the descending or transverse colon. It gradually moves in the direction of the path of the colon; it may be made to retrace its path by forced enemata. It hardens during the attacks of pain and may disappear between. Right iliac fossa appears peculiarly empty.

RECTAL TUMOUR.—A mass which feels like a soft os uteri may be felt per anum, or may protrude from the bowel. This occurs in about one-third of the cases, especially in children, in whom it may appear on the second day of the attack. It occurs only in colic and ileocæcal varieties. From three to eight inches may protrude, and the ileocæcal valve and appendix orifice may be recognized at the apex.

The Course of the Case.—The acuteness is determined by: (1) The site of the lesion—the higher being the more acute; (2) The youth of the patient. Ultra-acute cases in infants under one year may die within twelve hours. Eighty per cent die within eight days.

Diagnosis should never be in doubt, but Henoch's purpura may give some difficulty.

Methods of Spontaneous Recovery.—(1) Natural reduction, especially after opium; (2) Spontaneous elimination of the intussusceptum. This occurs most frequently in adults. It affects about 30 per cent of the cases, and of these perhaps only 10 per cent recover; the remainder die from perforation, ulceration, or hæmorrhage of the bowel. The time of separation varies from three days to six months, occurring in most cases at the end of the first month. It occurs more frequently in the enteric than in the other varieties.

Chronic Intussusception.—Usually of the colo-colic variety, and in adults rather than children. May last from a month to a year, the course being very irregular, and vomiting, pain, and diarrhœa all being variable in occurrence Blood-stained motions with mucus occur in about half the cases. Visible peristalsis is well marked An abdominal tumour is felt in about half the cases, and felt per rectum in about one-third.

Treatment.—

REDUCTION BY ENEMATA is now never employed.

OPERATION IN REDUCIBLE CASES.—Median incision. The tumour is brought into the wound. The apex is squeezed in an ascending direction through the sheath. It is useless and dangerous to drag on the entering layer at the neck. The greatest difficulty is in the final reduction of the œdematous apex. The appendix may be brought through the parietes and cut off to form a stoma through which feeding can take place. This also anchors the ileum and prevents recurrence. Or a simple stitch may be placed in the last part of the mesentery, fixing it to the parietes.

Intussusception—Treatment, *continued.*

OPERATION IN IRREDUCIBLE CASES.—

Jessets' Operation.—If the sheath is healthy, first tuck a little more intussusceptum into the sheath, then sew the two together by continuous stitch; open the sheath by a longitudinal opening and withdraw intussusceptum; cut it off transversely, sewing together the two layers in stump.

Total Resection of the intussusception is indicated only when the sheath is gangrenous This carries a high mortality.

V. OBSTRUCTION DUE TO FOREIGN BODIES

1. Swallowed Foreign Bodies of an indigestible nature.—

a Metal and other hard substances swallowed by accident, e.g., tooth-plates, or by design by lunatics or showmen. These usually pass per anum, but they may become lodged anywhere, especially in the cæcum, and cause obstruction. More commonly they perforate the peritoneum, the parietes, or some other viscus, e g , the bladder.

b. Sharp foreign bodies, e.g., pins or nails. These may accumulate in the stomach or duodenum, or perforate the alimentary canal and wander to distant parts without causing symptoms.

c Accumulated masses of débris. Hair, thread, fibres, fruit-stones or skins. Hair-balls may be found in lunatics and hysterical women who chew their own hair

2. Gall-stones.—Gall-stones which cause obstruction always enter the gut by ulcerating their way from the gall-bladder into the duodenum. Less often they enter the hepatic flexure of the colon in the same way, but then very rarely cause obstruction. They often become added to in the gut by deposits of magnesia, or carbonate of lime. Impaction occurs in the lower ileum, or the duodenum or jejunum. Patients are generally women, average age seventy.

3. Enteroliths, or stones formed in the intestine.—These may be: (*a*) Phosphates, which are probably deposited from catarrhal secretions; (*b*) Mineral deposits from medicines, e g., magnesia, bismuth, salol, etc. Actual obstruction by any of them is very rare, and then usually occurs from impaction in the lower ileum.

Clinical Aspects of Intestinal Obstruction due to Gall-stones.—

Sex and Age.—Three-quarters of the patients are females, usually elderly.

History.—Previous attacks of biliary colic or of local peritonitis round gall-bladder

Onset is abrupt Pain is severe but intermittent.

Vomiting is early, incessant, copious, and often stercoraceous.

Constipation and Collapse are in proportion to the acuteness of the attack.

The majority of cases are acute, but subacute and chronic cases occur.

UNUSUAL CASES OF GALL-STONE OBSTRUCTION.—Rarely after an acute onset, sudden relief may be caused by the stone passing the ileocæcal valve. A few weeks or months later the stone may pass the anus. Or there may be several successive attacks of subacute obstruction at varying intervals. Or the case may present all the characters of chronic obstruction, such as that due to stricture.

Clinical Aspect of Obstruction due to Other Foreign Bodies.—This is similar to that due to gall-stones, except that when due to enteroliths the case is decidedly chronic, and often ends by the natural passage of the concretion.

Course and Prognosis.—65 per cent die. Death or recovery at about the end of a week.

Treatment.—The abdomen is opened in the midline. The bowel is incised longitudinally rather above the foreign body. The latter is removed, and the wound sewn up.

VI. STRICTURE OF THE INTESTINE

1. Cicatricial Stricture.—Produced by the healing of an ulcer.

A. INFLAMMATORY ULCERATION.—(*a*) Duodenal ulcer. Very rarely causes stricture, and then the symptoms are rather those of dilated stomach. (*b*) Tuberculous ulcer. The strictures may be multiple; they are most often in the last part of the ileum or at the ileocæcal valve. (*c*) Typhoid ulcers very rarely cause stricture. (*d*) Syphilitic ulcers: very rare cause of stricture, except in the rectum. (*e*) Dysenteric ulcers cause stricture in the rectum, sigmoid flexure, descending colon, splenic and hepatic flexures. Taking the above all together, the large gut is affected by stricture six times as often as the small.

B. AFTER HERNIA.—Follows ulceration or limited gangrene of the gut due to strangulation. The symptoms follow from one month to several years after reduction. The ileum is the part generally affected.

C. AFTER INJURY.—This may be: (*a*) Surgical operations, e g., anastomosis, when mechanical methods like Murphy's buttons are a more frequent cause than simple suture. (*b*) After a blow on the abdomen which has produced a partial laceration of the gut. Here adhesions generally form and help in the production of obstruction. (*c*) Foreign bodies, e.g., gall-stones or tooth-plates.

D. PERIDIVERTICULITIS (*see* Chap. XXXIX). A dense contracting fibrous mass is formed round diverticula of the colon which have been the seat of chronic inflammatory changes. Commonest in the pelvic colon

2. Cancerous Stricture (*see also* CARCINOMA OF THE COLON, Chap. XXXIX).—Carcinoma is always cylindrical-celled. It may produce obstruction in various ways · (*a*) Most commonly by a limited annular band, like a narrow tape, tied round the gut (*Fig.* 165); (*b*) By a fungating mass filling up the lumen of the gut; (*c*) By producing a kinking; (*b*) Very rarely by causing an intussusception.

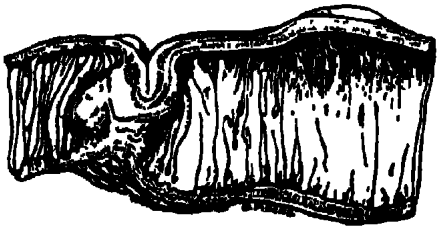


Fig. 165.—Cancer of the colon with almost complete obstruction, shown in longitudinal section. From the outside it looks as though a piece of string had been tied round the gut; hence the term 'string carcinoma'.

Cancerous Stricture of the Intestine, *continued.*

Dense and extreme contraction of the growth, fungation towards the lumen, ulceration at or above the growth, and colloid degeneration, may be found in the affected part. Adhesions to, or invasion of, surrounding structures may occur, but are much later and more exceptional than in malignant growths elsewhere. Metastatic growths occur in the liver, glands, and lungs, but these are late and often quite absent, even in advanced cases. Carcinoma may occur at any point, but is commonest at the sigmoid flexure, the descending colon, the splenic and hepatic flexures, and the ileocæcal valve.

3. Congenital Strictures.—These are all very rare compared with 1 and 2. They occur most frequently in the ileum, a few feet from the ileocæcal valve, where they may be due to an over-obliteration of the vitelline duct; then the duodenum or jejunum. They may amount to an absolute discontinuity of the gut narrowing to a fibrous cord, a membranous diaphragm, or any degree of stricture. In the colon a very few cases have been recorded. Symptoms of pyloric obstruction in the duodenal cases, or of intestinal obstruction in the others, occur in severe cases, and are fatal a few days after birth. They may remain latent for a few years or throughout life.

Locality of the Non-Congenital Strictures.—The colon, and especially the sigmoid flexure, is much more commonly affected than the small gut, 60 per cent of all cases of stricture being in the sigmoid.

In the small intestine, 60 per cent are caused by cicatrization—38 per cent from ulcers, 15 per cent from hernia, 7 per cent from trauma—40 per cent by cancer

In the large intestine (excluding the rectum), 65 per cent are caused by cancer, 35 per cent by cicatrization.

The Mechanical Conditions and Effects of a Stricture.—A stricture will not cause absolute obstruction so long as the contents of the gut at this point are fluid. Hence fatal small-gut strictures are much tighter than those in the large gut. Actual obstruction may be caused by: (1) Blocking by fæcal material or fruit débris; (2) The production of kinking or volvulus.

Treatment.—

FEEDING should be by bland diet which leaves but little residue. Fruit, nuts, vegetables, etc., especially to be avoided

APERIENTS must be used with caution. Salines are generally of most use, and in cases where the small gut is involved. They must, of course, never be used when obstruction is absolute.

OPERATION.—

FOR NON-MALIGNANT STRICTURE an enteroplasty or resection is indicated.

FOR LONG STRICTURE BURIED IN ADHESIONS a short-circuit will have to be done.

FOR MALIGNANT STRICTURES.—EXCISION is the ideal. It ought to be preceded by a short-circuiting operation or colostomy if the patient's condition is very bad, or if great distension exists above the growth.

In inoperable growths the choice lies between A SHORT-CIRCUITING OPERATION, which should always be done if possible, and an inguinal COLOSTOMY or cæcostomy, the lumbar colostomy having been almost abandoned.

VII. OTHER NEW GROWTHS OF THE BOWEL

These are all rare and seldom cause obstruction.

1. POLYPI may be adenomata, fibromata, fibromyomata, or submucous lipomata; rarely cystic of congenital origin. They occur most commonly in the rectum (80 per cent), and next in the ileum or colon. They are commonly multiple, and when small cause no symptoms. When they cause intestinal obstruction, this is similar to that caused by stricture. They may separate at their pedicles and pass per anum.
2. LYMPHADENOMA AND LYMPHOSARCOMA occur as submucous growths.

VIII. PRESSURE OUTSIDE THE BOWEL

Causes.—

UTERINE.—Large retroverted uterus, especially with: Fibroids or pregnancy—Extra-uterine gestation—Pelvic cellulitis—Cancer of the uterus.

OVARIAN.—Growths of any kind, but especially solid or malignant.

VESICAL.—New growths, notably cancer.

MESENTERIC.—New growths, especially cysts, subperitoneal tumours, or hydatid cysts.

RENAL.—New growths. Rarely obstruction is caused by the pedicle of a movable kidney.

PANCREATIC —Cancer, or rarely cysts.

SPLENIC.—Large and movable spleens.

HEPATIC.—New growths from the liver or bile-ducts, or glands in the portal fissure.

Regions Affected.—Rectum, 60 per cent; descending and pelvic colon, 12 per cent; lower ileum, 10 per cent, duodenum, 7 per cent; other parts, 11 per cent.

Clinical Characters.—These are of all varieties, viz., acute, intermittent, chronic, or chronic ending in acute obstruction.

IX. FÆCAL ACCUMULATION

(*See also* FUNCTIONAL DISEASES OF THE COLON, Chap XXXIX.)

Causes.—

1. DEFECTIVE EXPULSIVE POWER, due to some nerve condition or general debility, possibly the over-use of aperients.
2. INHIBITION OF DEFÆCATION.—Resulting from piles, fissure, or any pain in the pelvis, inflamed appendix or ovary, careless habits.
3. A LARGE QUANTITY OF INDIGESTIBLE FOOD and small quantity of liquids.
4. SOME ANATOMICAL ABNORMALITY.—Dilatation and prolapse of the cæcum, sigmoid, or transverse colon. Exaggerated sacculi. Adhesions round various parts of the colon, especially near the flexures. Thickening, contraction, and kinking by parts of the mesocolon.

Physical Characters and results on the bowel.—It occurs in the rectum, sigmoid flexure, or cæcum, in this order, or in the whole large gut. The accumulations are of two kinds: isolated masses of stony hardness from

Fæcal Accumulation—Physical Characters, ***continued.***

which nearly all moisture has been absorbed, and more ordinary fæcal masses. They are often covered with mucus or shreds of mucous membrane. The colon becomes dilated, elongated, and hypertrophied; it may measure 6 in. in diameter; its folds are exaggerated, and its sacculi unduly prominent. Stercoral ulcers are frequent, and may lead to perforative peritonitis.

Clinical Characters.—

COMMONER IN WOMEN THAN MEN. After middle age and in hypochondriasis.

CONSTIPATION is habitual, but attacks of spurious or catarrhal diarrhœa sometimes occur. Bowels may not act for weeks, or even months.

DIGESTIVE DISTURBANCES —Poor appetite, offensive breath, foul tongue, eructations, flatulence, and dyspepsia

MENTAL CHANGES.—Headache, vertigo, apathy, languor, hypochondriasis, or even insanity.

SKIN is dull, dark, greasy, and of an unpleasant odour; the conjunctivæ are dull and discoloured

TEMPERATURE rises occasionally, especially after aperients.

ABDOMINAL DISTENSION may become marked, and produce dyspnœa and palpitation Visible peristalsis is unusual.

OBSTRUCTIVE ATTACKS, with great pain and vomiting, occur from time to time, and may prove fatal.

TUMOUR is felt in the course of the large gut, and is formed by masses of fæces Most common situation is the cæcum, then the sigmoid. Very rarely it is doughy It is often tender from stercoral ulceration.

DEATH is rare, as the cases yield to treatment, but acute obstruction, perforation, septicæmia, or cardiac failure may cause it

Treatment.—

1. DRUGS, especially saline aperients. The more powerful cathartics usually do more harm than good.
2. LARGE DAILY ENEMATA —(*a*) Soap and water, (*b*) Olive oil, given a pint over night and retained till next day, when it is followed by a simple enema Manual removal of fæces from rectum.
3. SYSTEMATIC EXERCISE and massage where practicable.
4. APPENDICOSTOMY.—The stump of the appendix sewn into an opening in the abdominal wall. Through it the whole large gut can be washed out daily. This lavage can be carried out by the patient.
5. ILEO-SIGMOIDOSTOMY and excision of the colon above the sigmoid, when all other remedies have failed.
6. SYMPATHECTOMY.—Excision of the lower lumbar sympathetic ganglia (*see* p. 155).

CHAPTER XXXVIII

HERNIA

Definition.—Protrusion of an internal viscus, or portion of a viscus, through an abnormal opening in the parietes.

Signs of Abdominal Hernia.—

1. SWELLING, generally situated in the inguinal, femoral, or umbilical region, or in site of a scar
2. AN IMPULSE is imparted to this swelling on straining. The impulse is of an expansile character. The swelling is not only pushed out; it also gets larger.
3. REPLACEMENT can be effected into the abdomen of the contents of the hernia (unless irreducible), and this is often sudden and gurgling. When the contents of the hernia reappear, the swelling re-forms from above downwards

NOTE —Signs 2 and 3, which prove the connexion between the contents of a swelling and the interior of the abdomen, cannot be obtained if the hernia is irreducible. and in this case the diagnosis is conjectural.

Causes.—

CONGENITAL.—Patency of the funicular process Late descent of the testis. Weakness of the parietes, with large rings Length of the mesentery Congenital apertures in the linea alba. Malformation of the umbilicus or of the diaphragm

ACQUIRED.—Violent exercise (by sudden increase in intra-abdominal pressure) Wearing tight belt, girth, or stays Anything which causes frequent straining· Bronchitis—Large prostate—Constipation—Phimosis Pregnancy and parturition. Fat or tumours in the abdomen. Slipping down of the mesenteric attachment. Senile atrophy of abdominal muscles

Structure of Hernia.—

SAC.—Peritoneum pulled down through aperture or present as a congenital defect (In early cases the sac can be pushed back.) In established cases the sac is fixed outside the parietes.

Inflammation produces (1) Thickening of the sac; (2) Adhesion of its contents; (3) Loculation—hour-glass contraction or hydrocele of the sac.

COVERINGS are formed by all the structures which originally cover the hernial orifice. Tend to become matted together at the hernial orifice.

CONTENTS.—

SMALL INTESTINE.—Enterocele.

OMENTUM.—Epiplocele, especially likely to become irreducible. May develop serous cysts.

LOOSE BODIES.—From detached tags of omentum or appendices epiploicæ.

Structure of Hernia—Contents, ***continued.***

COLON OR CÆCUM.—Rare, but found in children.

BLADDER.—Either the fundus or a lateral saccule. In very large hernias.

ANY VISCUS except the pancreas may descend into a hernial sac in various conditions of enteroptosis.

INGUINAL HERNIA

Varieties.—

1. OBLIQUE OR INDIRECT—ACQUIRED—CONGENITAL (vaginal or funicular)—INFANTILE—INTERSTITIAL.
2. DIRECT (always acquired).—INTERNAL or EXTERNAL.

Orifice of Protrusion from the abdomen.—

OBLIQUE hernias all go through the internal abdominal ring. External to the deep epigastric artery.

DIRECT hernias leave the abdomen internal to the deep epigastric artery, i e., in Hesselbach's triangle, between the outer border of the rectus and the epigastric artery.

EXTERNAL DIRECT hernias leave the abdomen between the epigastric artery and the obliterated hypogastric

INTERNAL DIRECT hernias between the hypogastric artery and outer border of the rectus

Coverings of an inguinal hernia. From without inwards :—

OBLIQUE VARIETIES —

1. Skin, fasciæ.
2 Intercolumnar fascia from the external oblique.
3. Cremasteric layer from the internal oblique.
4. Infundibuliform fascia from the transversalis fascia.
5 Subserous fatty tissue
6. Peritoneum forming the sac

EXTERNAL DIRECT HERNIA.—As above, except the transversalis fascia instead of the infundibuliform

INTERNAL DIRECT HERNIA is covered by transversalis fascia and conjoined tendon, instead of the infundibuliform fascia and cremasteric layer

INFANTILE HERNIA has two additional peritoneal coverings next to the sac.

Degree of an inguinal hernia.—

COMPLETE.—Emerges through the external abdominal ring and descends into the scrotum or labium.

INCOMPLETE or bubonocele—Lies between the internal and external rings under the aponeurosis of the external oblique.

Acquired Oblique Inguinal Hernia.—Sac formed by abdominal peritoneum Develops slowly and is often incomplete. In early stages is above and distinct from the testis In later stages it lies in front of the testis. Structures of the cord are spread out over the sac.

Congenital Inguinal Hernia.—Sac formed by funicular process. May arise in infancy. May arise in adolescence (in this case it forms rapidly and soon becomes complete). Commoner on the right side. Very liable to strangulation. Structures of the cord are intimately adherent to the sac.

FUNICULAR VARIETY.—Sac lies above and distinct from tunica vaginalis and testis (*Fig.* 166, A).

VAGINAL VARIETY.—Sac is continuous with the tunica vaginalis (*Fig.* 166, B). Testis is enveloped by the hernia.

Infantile Inguinal Hernia consists of an infantile hydrocele and an inguinal hernia. The funicular process is obliterated at the internal ring, but patent thence downwards, and opens into the tunica vaginalis. The hernial sac is: (1) Pushed down behind the hydrocele sac (*Fig.* 166, C); or (2) Invaginated into it—the encysted variety (*Fig.* 166, D). Thus two layers of peritoneum, with fluid between them, cover the sac.

Direct Inguinal Hernia is always acquired. Generally in late life. Caused by a laxity and weakness of the internal oblique and transversalis muscles. Comes straight through the abdominal wall. Internal and external orifices in the abdominal wall are large and opposite one another. Epigastric artery runs on its outer side. Often remains small and incomplete.

Much more difficult to retain by truss or cure by operation than the oblique varieties. Because of: (1) Large size of the orifice; (2) Laxity of the abdominal muscles; (3) Directness of the descent

The cord is distinct from and to the outer side of the sac.

Interstitial Hernia.—A congenital oblique hernia in which the sac has an extra diverticulum. This lies:—

1. Between the peritoneum and muscles in front of the bladder, or in the iliac fossa—intraparietal variety; or
2. Between the external and internal oblique muscles parallel to the outer half of Poupart's ligament—interparietal variety (*Fig.* 167); or
3. Between the muscles and skin, along the front of Poupart's ligament: generally associated with retained testis—extraparietal variety.

Especially liable to give rise to difficulty in reduction by taxis.

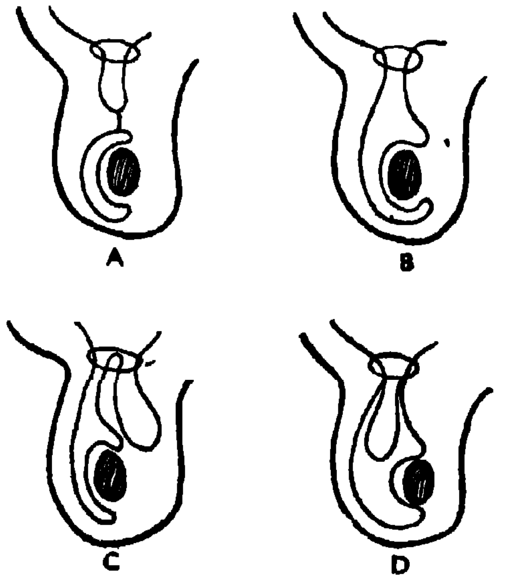


Fig. 166.—Inguinal hernia. Varieties of sac. A, Congenital funicular; B, Congenital vaginal; C, D, Two varieties of infantile (the funicular process should be shown closed at the internal abdominal ring).

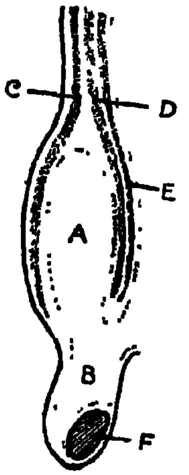


Fig. 167.—Interstitial hernia. A, Interstitial sac lying between muscle layers; B, Ordinary hernial sac extending down into tunica vaginalis; C, External oblique; D, Internal oblique; E, Transversalis; F, Testicle.

Inguinal Hernia, ***continued.***

Signs of an uncomplicated hernia :—

ROUNDED SWELLING in the groin above Poupart's ligament or extending into the scrotum or labium.

IMPULSE on coughing or straining. The swelling comes down farther and also increases in size—expansile impulse.

CONTENTS CAN BE REDUCED into the abdomen: Upwards and outwards in indirect varieties. Directly backwards in direct varieties. Reappears from above downwards. Gurgling generally accompanies reduction.

RELATIONS.—Poupart's ligament and groin fold lie below Pubic spine is below and on outer side in complete varieties Inguinal canal is occupied by the sac and its contents.

Diagnosis of Incomplete Inguinal Hernia from :—

RETAINED TESTIS.—Testicular sensation on pressure. Absence of testis from the scrotum. Impulse is only pushing, not expansile Upper limit of the swelling can be felt

SWELLINGS OF THE CORD —Hydrocele—Lipoma—Hæmatocele. Upper limit can often be felt. Reduction is impossible or very gradual. No expansile impulse Traction on testis pulls the swelling down

FEMORAL HERNIA.—In this the swelling appears first below Poupart's ligament. The inguinal canal has only the cord in it. The neck may be felt to be deep to Poupart's ligament.

ABSCESS in the groin, especially a chronic psoas or iliac A large intra-abdominal swelling exists Signs of primary disease. Outline is indefinite. Fluctuation exists.

GLANDS IN THE GROIN —Non-reducible. No expansile impulse. Tender, with indefinite outline Primary source of infection present. Solid feeling.

Diagnosis of Complete Inguinal Hernia from :—

FEMORAL HERNIA.—Sac lies below and outside pubic spine. Reduction occurs downwards and then upwards. Distinguished by other signs mentioned above.

VARICOCELE —Feels like a bag of worms. Does not generally extend into the inguinal canal Disappears on lying down Reappears from below upwards.

CONGENITAL HYDROCELE —Generally in infants Elastic or fluctuating. Translucent Reduction is very gradual No impulse as a rule.

Diagnosis of Inguinal Hernia when Irreducible or Inflamed.—

WHEN INCOMPLETE:—

From GLANDULAR SWELLINGS may be impossible. Primary source of infection. Gradual development. Normal inguinal canal.

ACUTE ABSCESS. Limits are outside the inguinal canal Primary cause, e.g., parametritis, can be found.

WHEN COMPLETE:—

From SOLID ENLARGEMENTS OF THE TESTIS.—The cord is thick in tubercle and new growth. Enlargement is slow and gradual. Feels solid and heavy. No impulse.

Treatment of Uncomplicated Inguinal Hernia.—

TRUSSES.—Trusses should not be prescribed when an operation is feasible.

In Infants.—May produce a cure in about one year. Should be rubber-covered. Should never be removed without supporting the inguinal region.

In Adults.—Can only support the rupture. Will never cure the hernia. Dangerous if it does not control the hernia. Hernia tends to become larger from constant truss pressure.

Measurement.—Round pelvis, midway between highest point of crest and top of the great trochanter, to meet over symphysis = size in inches.

Spring.—Must press backwards and a little upwards. Requires renewal two or three times yearly.

Adjustment.—Only apply after hernial contents have been reduced. Pad should be over the inguinal canal and not on the bones. Leg strap is necessary to keep hernia from coming under it May generally be left off at night.

Common Modifications.—(1) Rat-tail truss: pad is produced into a tongue process attached to leg-strap (2) Forked tongue truss: pad has two processes attaching it to leg-piece and to the cross-strap. Both useful for large scrotal hernias in which external ring is very large

RADICAL OPERATION —

Indications.—Healthy adults. Active occupation.

Contra-indications —Infants under one year, in whom a truss often cures. Senility. Lax atonic muscles. Enteroptosis Existence of chronic bronchitis, enlarged prostate, any cause of straining.

Doubtful Cases —Direct inguinal hernias—because the muscles are lax or torn Large hernias of old standing. Large irreducible hernias in old men.

Method.—Radical cure.

Incision half an inch above inner half of Poupart's ligament (a curved incision turning down a flap of skin is better).

Slit up the external oblique from the external to the internal ring half an inch above Poupart's ligament.

Define the sac and separate it from the structures of the cord. Separate adhesions and remove redundant omentum. Empty the sac after opening it. Tie the neck of the sac (after transfixing it) flush with the peritoneum Cut off the sac, and sew the stump to the deep surface of the parietes

In young healthy adults with good musculature herniotomy or removal of the sac is sufficient. Advantageous not to divide the external ring if not large, and to disturb the cremaster as little as possible.

If there is a definite weakness of the fascia transversalis, it is advisable to plicate this with catgut sutures.

If a repair is performed a ' muscle slide ' operation gives the best results. Anterior sheath of rectus is incised by a curved incision as shown in *Fig.* 168, A, and dissected off the rectus abdominis, so allowing the conjoined muscles to be sutured to Poupart's ligament without tension (Tanner's operation)

Occasionally Bassini's operation (*Fig.* 169) is used.

In large hernias the muscles are sewn together by a strip of fascia cut from the aponeurosis of the external oblique or by a fascial graft cut from the fascia lata (Gallie). Floss silk is also used.

Inguinal Hernia—Treatment, ***continued.***

INJECTION METHODS.—

PRINCIPLE.—To effect cure by causing sclerosis outside the sac. The sclerosing fluid is injected in the inguinal region outside the sac.

APPLICABILITY.—Only reducible inguinal herniæ which can be controlled by a truss are suitable.

N.B.—These are the ones most satisfactorily cured by an operation.

METHOD.—The hernia must never be allowed to come down during the cure, i.e., truss must be worn day and night during course of injections for three months.

COMMENT.—Not popular in this country—the method is only suitable for the 'easy' hernia which presents no problem. For the problem cases, which form the crux of hernia surgery, the method is unsuitable. While an operation is avoided, the method savours of 'hit and miss' and is not always reliable.

FEMORAL HERNIA

Aetiology.—Before puberty both sexes equally, but rarely, affected. Commoner in women after puberty, because of the increased width of the pelvis opening out the crural canal. Inguinal hernia is still the more common hernia in women.

Anatomy.—Emerges through the crural canal. Crural canal is innermost part of the femoral sheath. Femoral sheath is formed by the junction of: (1) The fascia transversalis in front of the vessels; (2) The fascia iliaca behind the vessels.

RELATIONS OF THE CRURAL CANAL and of the neck of a femoral hernia.—

IN FRONT.—Transverse thickened part of the femoral sheath—deep crural arch. Poupart's ligament—superficial crural arch. Iliac portion of fascia lata and cribriform fascia.

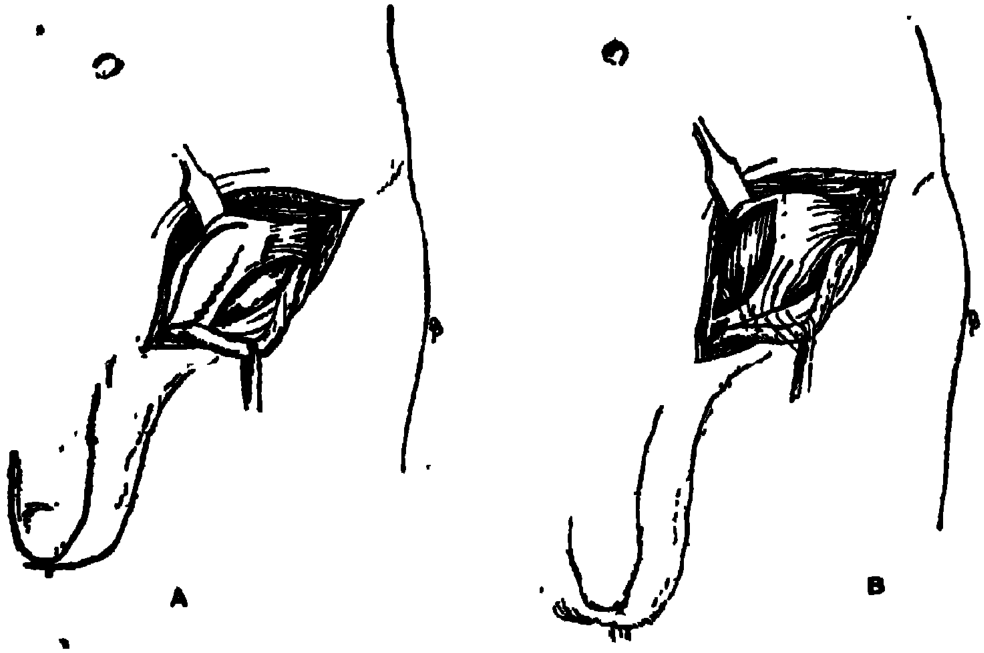


Fig. 168.—Tanner's 'muscle slide' operation.

On Inner Side.—Gimbernat's ligament.

On Outer Side.—Femoral vein.

Behind.—Os pubis and Cooper's ligament. Pectineus muscle covered by pubic portion of the fascia lata.

Above it is closed by subserous tissue—the septum crurale.

Below it abuts against the cribriform fascia closing in the saphenous opening of the fascia lata.

COVERINGS.—

1. Skin and fascia, etc.
2. Cribriform fascia.
3. Femoral sheath (transversalis fascia).
4. Subserous tissue (septum crurale).
5. Peritoneum.

NECK.—Situated at the crural ring.

In Front.—Poupart's ligament and inguinal canal with contents.

Inner Side.—Gimbernat's ligament

Behind.—Pubic part of fascia lata covering os pubis.

Outer Side.—Femoral vein.

RELATIONS TO VESSELS.—Femoral vein on outer side. Long saphenous vein enters below it Epigastric artery to its outer side.

Obturator artery is given off as branch of the deep epigastric in one in four cases. Runs on the outer side of the sac, between it and the femoral vein, generally. Runs on the inner side of the sac by Gimbernat's ligament in one in seventy-five cases. In these only is there much danger of its division in herniotomy.

DIRECTION OF GROWTH —After emerging from the saphenous opening, the hernia is turned upwards over Poupart's ligament by the attachment of the deep layer of the superficial fascia to the fascia lata along a horizontal line level with pubic spine.

Contents.—Small intestine (ileum)—generally. Omentum—sometimes. Large intestine when left-sided—rare. Ovary or Fallopian tube—occasionally.

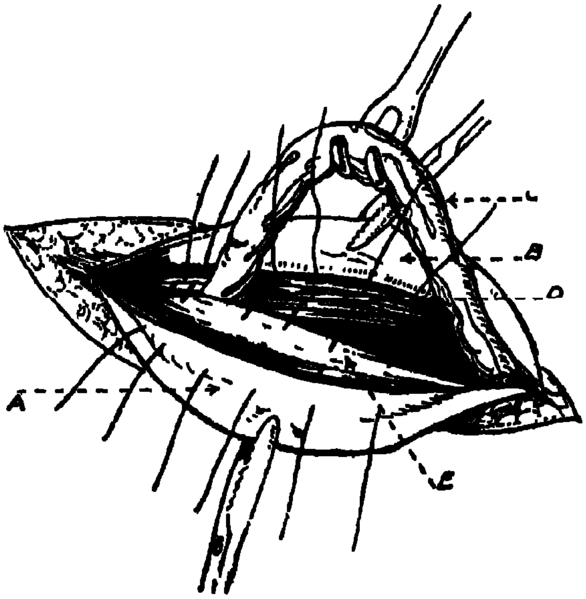


Fig. 169.—Bassini's operation for radical cure of inguinal hernia. A, Aponeurosis of external oblique, lower portion; B, Upper part of the same; C, Spermatic cord, D, Conjoined tendon, being sutured to E, Deep surface of Poupart's ligament.

Femoral Hernia, *continued.*

Signs.—Swelling below inner end of Poupart's ligament. Reducible with a gurgle. Expansile impulse on coughing. Neck lies to the outer side of and below pubic spine; lies behind Poupart's ligament and inguinal canal. When it rises above Poupart's ligament it obscures the fold of the groin, and lies superficial to the inguinal canal.

Diagnosis.—May be difficult in fat women. when hernia is irreducible.

INGUINAL HERNIA and INFLAMED LYMPH-GLAND (*see* p. 438).

LIPOMA.—Long history and stationary size

PSOAS ABSCESS.—Relation to iliac swelling. Descends behind the femoral sheath.

SAPHENOUS VARIX.—Veins below are generally varicose. Often fills after emptying in spite of pressure over crural canal.

Treatment.—

TRUSS is less satisfactory than in inguinal hernia, because it presses on the vessels, and because the pad lies over the pubic bone, where pressure causes pain and rubbing. General opinion is now never to prescribe a truss.

OPERATION is less satisfactory because it is difficult to close the crural ring.

METHODS.—Vertical incision Isolation of sac. Open sac and empty. Tie neck of the sac high up Then, either:—

1. Lotheisen's operation: Make an incision above and parallel to Poupart's ligament and seperate the fibres of the external oblique aponeurosis in the line of their fibres for length of incision The sac is withdrawn through the crural canal after separation below, ligatured as high up as possible, and excised. The conjoint tendon

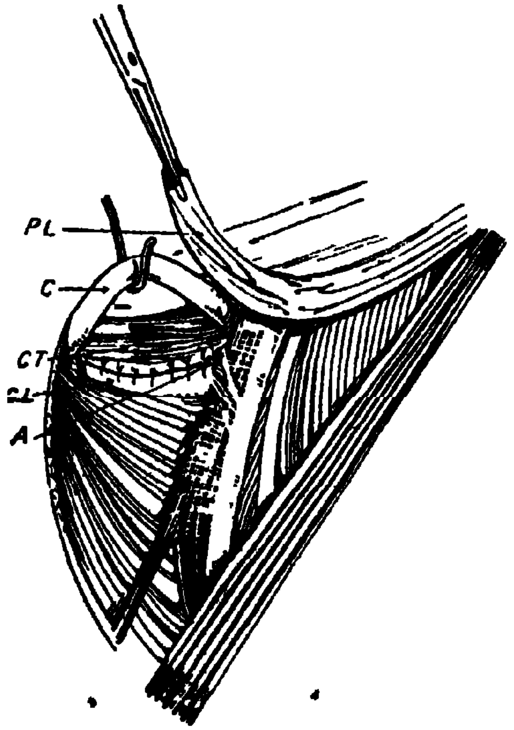


Fig. 170.—Operation for radical cure of femoral hernia. Obliteration of femoral ring and canal by suture of conjoined tendon to Cooper's ligament and pectineal fascia. P.L., Poupart's ligament; C, Cord; C-T, Conjoined tendon; C.L, Cooper's ligament, A, Junction between conjoined tendon, femoral sheath, and Cooper's ligament. Poupart's ligament shown detached, and operation shown from below for clarity.

and internal oblique are sutured to Cooper's ligament (along the horizontal ramus of the pubis). The slit in the external oblique is then repaired.

2. Divide pubic attachment of Poupart's ligament and then perform Lotheisen's operation (*Fig* 170).

UMBILICAL HERNIA

Varieties.—

CONGENITAL.—Hernia protrudes into base of umbilical cord. Gut may be tied and cut with the cord. Very rare.

INFANTILE.—Hernia through a stretched umbilical cicatrix. Tends to spontaneous recovery. Treated by a binder.

ACQUIRED —Hernia through linea alba above or below the navel. Generally in stout multiparous women. This is a para-umbilical hernia. Sac is very thin and lobulated Prone to complications, viz , obstruction and strangulation. Contents, which generally include part of transverse colon and omentum, are very adherent. Much fat is developed: (1) Outside sac wall; (2) In omental contents

Treatment.—These herniæ can seldom be treated by a truss as most cases are irreducible, and in the few reducible cases no truss can be made to control the neck of the sac In cases unfit to stand operation the bulk of the hernia may be received in a support

METHOD OF OPERATION —

IN SMALL HERNIÆ —Vertical incision Open sac. Remove omentum and separate adhesions Isolate and cut away sac. Cut margins of neck of sac so as to open rectal sheaths. Sew posterior sheaths of recti together Sew recti together. Sew anterior rectal sheaths together.

IN LARGE HERNIÆ, Mayo's operation is best A transverse incision is made around the umbilicus and hernia, and is deepened to the base of the hernia. The sac is opened at its neck, where usually no adhesions exist. The contents of the sac are reduced and the sac excised The peritoneum is separated for a short distance around the margins of the gap in the aponeurosis and is sutured with a purse-string suture The aponeurosis of the abdominal wall is then overlapped by mattress sutures so that one edge overlaps the other transversely by one or two inches The edge of the superficial flap is then sutured to surface of underneath flap.

OTHER VARIETIES OF HERNIA

Ventral Hernia.—

IN LINEA ALBA ABOVE NAVEL —Generally a protusion of subserous fat only. Small in size, but very painful

TREAT by removal of fat and sewing up hole in linea alba.

IN LINEA ALBA BELOW NAVEL —After parturition. Tends to spontaneous recovery if supported by a belt

In bad cases only, OPERATE. Sew edge of one rectus to the deep surface of the other. Sew edge of the other rectus to the superficial surface of the first.

Post-operative.—Especially after suppurating appendicitis.

OPERATE on all young patients. Sew abdominal layers together one by one. Use of 'living fascia suture' (e.g., fascia lata) makes relapse much less likely.

PATHOLOGICAL CONDITIONS OF HERNIA

Varieties.—

1. IRREDUCIBILITY.
2. INFLAMMATION.—Involves 1.
3. OBSTRUCTION.—Involves 1.
4. STRANGULATION.—Involves 1, 2, and 3.

I. IRREDUCIBLE HERNIA

Definition.—Contents of a hernia cannot be returned into the abdomen.

Causes.—

ADHESIONS OF THE CONTENTS TO THE SAC.

ADHESIONS OF THE CONTENTS TO EACH OTHER, so that a bulky mass larger than the neck of the sack is formed.

ADHESIONS OF THE WALLS OF THE SAC TO EACH OTHER, forming bands or constrictions. Follows attacks of inflammation. Occurs during obstruction and strangulation.

PRESENCE OF OMENTUM in the sac From adhesions, fat accumulation, or cyst formation.

Occurrence.—In order of frequency—in umbilical, femoral, and large scrotal herniæ

Signs.—A swelling at one of the hernial orifices Gurgling may be felt and heard Portion of the contents can often be reduced.

Diagnosis.—From the following (*see above*)· Inflamed glands—Lipoma of parietes or cord—Abscess (e g , psoas abscess)

Dangers.—Obstruction—Strangulation.

Treatment.—

FIRM PRESSURE.—In bed, by bandages and ice-bags

OPERATION for radical cure

Contra-indications for operation· Old patients with no symptoms. Very large hernia.

SUPPORT by a bag truss.

II. INFLAMMATION OF A HERNIA

Definition.—Inflammation of the peritoneal sac and of the contents of a hernia.

Causes.—Injury, e.g , by taxis Escape of bacteria through the gut wall. Strangulation.

Anatomy.—An exudation from the sac wall and from the peritoneum covering the contents. Nature of the exudation is serous, plastic, or purulent.

Signs.—Those of external inflammation. Heat, redness, tenderness, and œdema over the hernia. Hernia is irreducible. Temperature rises. Some constipation and vomiting.

Results.—Abscess forms and bursts externally. Strangulation may supervene. Irreducibility generally remains.

Treatment.—Rest in bed, with fomentations. Incision and drainage if suppuration occurs. Radical operation cannot be done until the septic inflammation is over.

III. OBSTRUCTION OF A HERNIA

Definition.—Lumen of the gut in a hernia is obstructed without strangulation of its coats.

Causes.—Presence of solid matter in the gut. Specially liable to occur in umbilical hernia, which often contains the transverse colon. Complicated matting together of the gut interferes with its peristaltic action.

Symptoms.—The hernia is irreducible and larger than usual. Constipation, vomiting, and colicky pains—occurring together in attacks lasting several hours or days.

Results.—Recovery from early attacks as a rule. Strangulation ends an attack ultimately.

Treatment.—Rest in bed with low diet. Pressure and an ice bag over the hernia. Copious enemata. Radical cure when the obstruction is over.

IV. STRANGULATED HERNIA

Definition.—Constriction of the contents of a hernia to such a degree as to stop the circulation in them.

Causes.—

a. Existence of a Tight Strangulating Band or constriction. The neck of the sac itself in congenital inguinal cases. The matted tissues at the internal abdominal ring in acquired inguinal cases. Gimbernat's ligament in femoral cases. Bands of adhesion inside the sac.

b. The Forcing Down of an Extra Portion of the viscera through the neck of the sac.

c. Adhesions and Kinking of coils of gut resulting from inflammation.

Pathology.—

a. THE GUT.—

First Stage.—Stasis of the circulation, with venous engorgement. Colour is dark red or purple. Great thickening of the gut wall from œdema. Distension of the lumen by gas. Escape of bacteria, viz., *Bacillus coli*, through the gut wall. Small ecchymoses may occur. Peritoneum is smooth and shiny. Vessels can be emptied by pressure and refill easily.

Second Stage.—Thrombosis. Peritoneum is covered by plastic lymph and has lost its lustre. Colour is a dark purple. Vessels cannot be emptied by pressure. Free passage of bacteria through the gut wall.

Third Stage.—Gangrene. Gut is a slate-grey or black. May be perforated, or gives way on handling.

At the Site of Strangulation.—A pressure ulcer occurs from the mucous surface, or the whole thickness of the gut sloughs at the point of greatest pressure. Bacteria escape through this. Perforation may occur before, during, or after reduction.

Above the Strangulation.—Distension of the gut from obstruction. Paralysis and peritonitis occur later if the condition has long continued.

b. OMENTUM.—Undergoes similar changes, but gangrene is much rarer.

Strangulated Hernia—Pathology, ***continued.***

c. SAC.—Undergoes the changes of peritonitis. Inflammation, with copious serous exudation. Exudation becomes bloodstained and offensive. Plastic lymph may be deposited on its surface. Sloughing from the virulence of bacterial action. Inflammation heralded by œdema of tissue outside the sac. Sac bursts and a fæcal fistula results.

Anatomical Varieties of Strangulated Hernia.—

SIMPLE ENTEROCELE.—The common form.

EPIPLOCELE.—Rarely strangulated unless bowel occurs in the hernia also.

PARTIAL ENTEROCELE (Richter's Hernia).—A part only of the circumference of a piece of gut is strangulated

HERNIA OF DIVERTICULUM (Littre's Hernia).—A blind diverticulum only is strangulated, e g., the appendix or Meckel's diverticulum.

Signs.—

AT FIRST —The hernia becomes hard or feels tense. Irreducible. No impulse on coughing Tender or painful History of recent enlargement.

LATER.—Skin becomes inflamed and œdematous. Dark colour with emphysema if patient survives.

THE ABDOMEN —Severe spasmodic pain referred to the umbilicus. Tenderness and rigidity markedly absent at first Distension and tympanites (from meteorism) late. Signs of peritonitis occur late.

Symptoms.—

SHOCK.—

MOST MARKED IN: Pure enterocele—Partial enterocele (Richter's hernia) —When a hernia becomes strangulated on its first occurrence (children congenital cases).

LEAST MARKED IN: Old-standing cases—Large herniæ—Cases preceded by irreducibility and inflammation—Epiplocele.

Pulse slow and weak, then rapid and irregular. Temperature subnormal, but rising with onset of inflammation. Faintness

PAIN —Referred to the region of the umbilicus. Also over the region of the hernia Occurs as spasms of colic supervening on a constant abdominal pain. Becomes less on the onset of gangrene.

VOMITING.—First of food. Later of bile and small intestine contents. Lastly of foul stercoraceous fluid.

CONSTIPATION.—Absolute from the first in most cases. Caused by: (*a*) Obstruction of the lumen of the gut; (*b*) Paralysis of the gut by interference with its nervous mechanism; (*c*) Peritonitis (at last).

Not present at first: In strangulation of the gut high up, when the lower bowel may empty itself.

Not present at all: In epiplocele, or any case when a viscus (e.g., ovary) other than gut forms the contents of the hernia. In partial enterocele (rare).

TOXÆMIA.—Occurs after the case has persisted some time. Shrunken face, hollow eyes, anxious expression. Temperature becomes again subnormal. Drowsiness, delirium, and coma.

LATENCY OF THE SYMPTOMS is often well marked and very important. Occurs especially in elderly patients, and in old-standing hernias. Shock may be absent entirely. Only symptoms present may be constipation, a little vomiting, and local tenderness over the hernia.

Gangrene of the gut in such cases may be signified by: Relief of pain in the abdomen. Relief of tension and tenderness in the hernia. Occurrence of œdema over the hernia.

N.B.—Gangrene of the gut may occur within a few hours of strangulation.

Treatment.—

TAXIS.—Should be used in the following conditions only, and only in inguinal hernia:—

1. When the hernia has only just appeared, e.g., in congenital hernia, when the contents come down suddenly and are at once strangulated.
2. When the hernia has been reducible a short time previously.
3. Within a few hours of the onset of strangulation.
4. For a short time (ten minutes).
5. With the utmost gentleness.
6. Under an anæsthetic, which may be prolonged for the operation of herniotomy.

DANGERS.—Except in the cases mentioned above, taxis is much more dangerous than operation, and is often the cause of a fatal result following an operation.

1. *It may damage the sac and its contents*, especially the gut. Causes œdema and swelling. Produces hæmorrhage into the sac and into the gut wall. May rupture the gut
2. *It may force inflamed or gangrenous gut back* into the abdomen. Hastens the transudation of bacteria through the gut wall. Hastens gangrene. Causes unnecessary suffering and delay. Makes the subsequent operation more difficult and dangerous.
3. *It may reduce the hernia without relieving symptoms*:—
   - *a.* By the reduction of a ruptured or gangrenous gut
   - *b.* By causing peritonitis from the reduction of septic contents into the abdomen.
   - *c.* By forcing the sac with its strangulated contents back into the abdomen—'*réduction en masse*'.
   - *d.* By forcing the hernial contents into a pouch leading out of the main sac, such as occurs in an interstitial hernia—'*réduction en bissac*'.
   - *e.* By forcing the contents through a rupture in the neck of the sac.
   - *f.* By reducing contents whose strangulation is due, not to the neck of the sac, but to a band or kinking of the contents themselves.

OPERATION without any unnecessary delay. Directly the diagnosis is clear or even probable. In all except the few cases when taxis is permissible and successful. The indications for taxis are very few and it is safer to operate on all cases of strangulated hernia.

DANGERS OF DELAY IN OPERATION.—

No tendency to natural recovery, except the very rare occurrence of a natural fæcal fistula.

Sepsis begins to occur with strangulation, by an escape of bacteria through the congested gut.

Gangrene of the gut may result in a few hours.

Strangulated Hernia—Treatment, ***continued.***

Paralysis of the gut, with incapacity for recovery, may result from prolonged distension.

The general condition of the patient becomes worse and not better from delay, because of: (1) Want of food; (2) Want of sleep; (3) Loss of fluid by vomiting; (4) Rapidly increasing septic absorption.

Operation for Relief of Strangulated Hernia.—

PRELIMINARIES.—Wash out the stomach if vomiting is severe. Anæsthetize by local, spinal, or intratracheal anæsthesia.

EXPOSURE OF HERNIAL CONTENTS.—Cut down to the sac. Open sac carefully and let out contained fluid.

DIVISION OF THE STRANGULATING AGENT.—Usually found at the neck of the sac. Occasionally in the body of the sac or in the contents. In oblique inguinal herniæ, cut upwards and inwards at the neck of the sac. In femoral hernia, cut inwards Gimbernat's ligament. In all others, cut any tense band that may be felt. Make the incision as small as possible: just sufficient to free the contents.

EXAMINATION OF CONTENTS.—First pull the contents down farther, so that the actual site of strangulation can be examined. Decide whether the contents, especially the gut, are viable.

SIGNS OF VIABILITY of the gut or other viscus —The peritoneum is smooth and shiny. Blood-vessels can be emptied by pressure, and refill.

SIGNS OF THE GUT NOT BEING VIABLE.—The peritoneum has lost its lustre May be covered by lymph. Colour is deep purple or slate. Vessels cannot be emptied by pressure.

TREATMENT OF THE GUT and other hernial contents.—

ANY LIMITED ULCER at the site of strangulation, or

ANY LIMITED PATCH OF GANGRENE, should be infolded by sewing the neighbouring healthy gut over it.

VIABLE GUT should be returned into the abdomen.

REDUNDANT OMENTUM should be ligatured and removed.

NON-VIABLE GUT should be excised, cutting well above and below the site of strangulation. Then perform immediate anastomosis of the healthy ends, except in the following · (1) when great distension exists, or (2) when the patient's condition is desperate, sew the ends together in part of their circumference, and tie in a tube at the remaining part, pushing the junction just within the abdomen.

RADICAL CURE should complete the operation, except when the patient's condition is grave, or when gangrene and sepsis are evident. In this case drain through the opening in the parietes.

AFTER-TREATMENT.—Enemata of turpentine and soap until natural evacuations occur. Continuous rectal infusion of saline fluid in cases with marked collapse or peritonitis. Avoid feeding by mouth until the bowels have acted.

Complications after Operation.—Intestinal obstruction from paralysis of the gut. Peritonitis. Acute enteritis, passage of blood and mucus.

Chapter XXXIX

DISEASES OF THE COLON

Anatomy and Physiology of the Colon.—

Length of Large Intestine.—Five feet in adults, i.e., one-fifth of the whole intestine, and about the same length as the individual's body height.

Subdivisions of Colon:—

Name	Begins	Ends	Length	Circumference
Cæcum	Blind end	Ileocæcal valve	7 cm.	28 cm.
Ascending colon	Ileocæcal valve	Liver	10 „	20 „
Hepatic flexure	Liver	Liver		
Transverse colon	Liver	Spleen	50 „	15 „
Splenic flexure	Spleen	Spleen		
Descending colon	Spleen	Left iliac crest	15 „	14 „
Iliac colon	Left iliac crest	Left psoas	10 „	
Pelvic colon	Left psoas	3rd sacral vertebra	50 „	17 „

These figures vary very much, especially as regards the transverse and pelvic colon.

Capacity of Large Intestine.—In adults it holds 3 to 5 pints (2 to 3 litres), i.e., about the same capacity as the whole of the small intestine.

Special Characters of the Colon.—(1) The outer longitudinal muscle is arranged as three distinct bands or tæniæ. This becomes rearranged as two bands at the lower part of the pelvic colon. (2) The sacculations which bulge out between the tæniæ. (3) The appendices epiploicæ. Fatty appendages which are best marked in fat subjects, in old age, and in the transverse and pelvic colon.

COMMON VARIATIONS.—

The Cæcum may have the Appendix: (1) Abruptly demarcated and attached behind and to the inner side—the adult type; (2) Abruptly demarcated and terminal—the infantile type; or (3) Terminal with no sharp demarcation—the fœtal type.

Position of the Cæcum.—(1) Normally in the right inguinal region; Often (2) in the right loin close under the liver, (3) hanging over the pelvic brim or actually in the true pelvis; Rarely (4) behind the umbilicus, or (5) in left iliac fossa.

The Hepatic Flexure may be absent, the ascending running obliquely into the transverse colon.

The Transverse Colon varies much in length and position. When very long its middle part may lie in the pelvis.

Pelvic Colon varies: (1) In length—from 12 cm. to 84 cm.; (2) In position—commonly lying in the pelvis, more rarely as a loop upwardly directed across the abdomen; (3) In the length of mesentery—the pelvic mesocolon may be absent, or be anything up to 25 cm. long.

Anatomy and Physiology of the Colon, ***continued.***

CONSTRICTIONS.—

SPLENIC FLEXURE is so much kinked by the attachment of the phrenicocolic ligament as to be the narrowest point

RETROCOLIC JUNCTION.—There is often an abrupt angle where the pelvic colon passes into the rectum, and at this point a thickening of the circular muscle forms a kind of sphincter.

THE ILEOCÆCAL VALVE forms a muscular sphincter under nervous and reflex control, which regulates the admission of chyme from small gut into large. As a mechanical valve it is incompetent; fluid can be forced by enemata into ileum.

TIME TAKEN BY CONTENTS IN PASSAGE OF INTESTINES.—
Small intestine, 4 hours; ascending colon, 1–3 hours; transverse colon, 2 hours; descending and pelvic colon, 16 hours.

MOVEMENTS OF THE COLON.—

PERISTALSIS, as in the small bowel, drives on the intestinal contents towards the anus. Not as frequent as in the small bowel; mass peristalsis occurs

ANTIPERISTALSIS is probably a normal movement in the proximal part of the colon, and it tends to drive the fluid contents of the transverse and ascending colon back towards the cæcum. In certain conditions this antiperistaltic contraction is probably exaggerated

DIGESTION IN THE COLON.—

WATER ABSORPTION.—The colon is capable of absorbing an unlimited quantity of water Normally it absorbs about 16 per cent of the water from the chyme of the small intestine This is its principal function.

FOOD ABSORPTION.—Proteins, fats, and carbohydrates are absorbed in such small quantities as to be negligible. When injected into the rectum, emulsified fat and carbohydrates are absorbed to some extent, and proteins very slightly.

PUTREFACTION.—Proteins and fats are decomposed and carbohydrates undergo fermentation in the colon under the influence of the bacteria.

BACTERIA IN THE INTESTINES.—

THE DUODENUM AND JEJUNUM are almost germ-free, and if the mouth and food are sterilized they may be kept so.

THE ILEUM contains an increasing number of bacteria towards its lower part.

THE CÆCUM contains the maximum number and variety of bacteria. It is rightly called the intestinal cesspool—into it the fluid contents of both small and large guts are emptied.

THE COLON contains many varieties both of bacilli and cocci. The *Bacillus coli* and the pyogenic cocci predominate. About one-third part by weight of dried fæces consists of bacteria.

FUNCTIONAL DISEASES OF THE COLON

There are many cases of extreme constipation unassociated with any gross disease of the colon; there are others—comparatively rare—in which the colon is lengthened, kinked, or dilated, as a primary congenital defect; and lastly, there are cases in which constipation and dilatation of the colon occur and increase *pari passu*, the one condition sometimes being the cause and sometimes the result of the other. For purposes of description it will be convenient to take the extreme type of idiopathic dilatation of the colon, although this is the rarest.

IDIOPATHIC DILATATION OF THE COLON

(Hirschsprung's Disease)

Aetiology.—

Sex.—Males always are more liable than females. Proportion is males : females :: 8 : 1.

Age.—The majority occur in infants or children under 10.

Cause.—Congenital mal-innervation of the colon probably accounts for the majority. In others, long-standing constipation may be the cause and the colonic dilatation the result. Overaction of the sympathetic nervous system. A similar condition may occur in the urinary bladder, viz., over-distension and imperfect emptying, in association with Hirschsprúng's disease.

Pathological Anatomy.—

Parts Affected.—The pelvic colon is alone affected in nearly 30 per cent, and with other parts in more than 60 per cent The whole colon in uniformly dilated in about 15 per cent.

Measurements.—An increase in length is frequent, but not invariable, and is less often found in the very young children than in those of older years. The girth is dilated so that the circumference is often as great as that of the patient's thigh The capacity is correspondingly increased, being as much as 7 pints in infants.

Kinking is necessitated in order to accommodate the voluminous gut. This is best marked in the transverse and pelvic portion, and especially at the junction of the colon and rectum.

Muscular Hypertrophy —The muscular wall is usually much thickened, and occasionally a long or short segment of the colon is tightly contracted. A thickness of 6 mm , or three times the normal, is common.

Inflammatory Changes in the peritoneum are conspicuously absent. Stercoral ulcers may occur in the lumen and lead to perforation.

Secondary or Pressure Changes.—Venous obstruction, with œdema of the legs. Scanty, albuminous urine. Diaphragm, heart, and lungs pushed upwards

Physical Signs.—

Abdomen is distended and globular, tympanitic to percussion, and showing no free fluid

Visible Peristalsis —Large coils of colon are disposed chiefly in a longitudinal direction, and these contract visibly and audibly.

Chest is contracted and short, being only 1/5 or 1/6 the length of the trunk, instead of the normal 2/5. The lower rib margins are everted.

Symptoms.—

Onset.—Gradual, being preceded by a variable latent period.

Constipation.—Varies very much. Usually it is conspicuous, the intervals between going to stool varying from one week to three months. Some, however, have regular daily motions, but this does not imply that there is no intestinal stasis.

Diarrhœa.—This may alternate with constipation and afford temporary relief to the distension.

Idiopathic Dilatation of the Colon—Symptoms, ***continued.***

Emaciation with Loss of Appetite occurs sooner or later in severe cases, and indicates that treatment must be undertaken without delay.

Nervous System.—In infants convulsions or tetany, in adults neurasthenia or melancholia.

Causes of Death.—Perforative peritonitis—Bronchitis or bronchopneumonia—Convulsions—Toxæmia.

Course.—The length of the disease varies from a few weeks to many years. The earlier the symptoms manifest themselves the more rapid is the course of the case.

Cases in Adults in which Constipation is the Primary Factor.—These differ from the above-described cases of idiopathic dilatation of the colon in the following respects:

Parts Affected.—The upper part of the colon, i.e., the cæcum and ascending and transverse portions, is most frequently involved, whereas in the congenital or idiopathic variety it is the pelvic colon.

Sex —Females greatly preponderate over males, in a proportion of 5 or 7 to 1, just the inverse proportion to that obtaining in the congenital disease.

Symptoms.—Constipation does not date from early life; distension of the abdomen is not marked, and visible peristalsis is not seen.

Treatment.—

Diet.—Easily digested food—milk, fish, eggs, and farinaceous food. Some cases react better to a coarse diet of brown bread, porridge, and vegetables, but such are exceptional.

Drugs.—Ordinary purgatives are worse than useless, especially when great distension exists. Tonics and stimulants, e.g., strychnine, aloes, and iron, may be of some use in conjunction with enemata, etc.

Enemata.—Whilst it is easy to inject large quantities of fluid per rectum, this may be retained or returned unchanged Olive oil (1 pint) given at night (and retained), followed by turpentine (1 oz.) in soapy water in the morning, is the most efficacious.

Mechanical Means.—Athletic exercises, abdominal massage, and electrical treatment will benefit the cases of slight degree. Defæcation should be performed in the squatting position, and tight corsets abandoned.

SURGICAL TREATMENT.—

Indications —Failure of diet, drugs, and enemata to afford permanent relief. Marked and increasing distension of abdomen. Visible intestinal peristalsis. Progressive emaciation. Recently it has been shown that high spinal anæsthesia will bring about a cure in some cases, and this may replace surgery in such cases. This treatment is coupled with the administration of acetylbetamethylcholine bromide and liquid paraffin with normal bowel habit.

Sympathectomy.—Operation of choice. All previous surgical measures have been replaced by operations on the sympathetic nervous system. Thus there is now no place for cæcostomy, appendicostomy, colostomy, colectomy, or short-circuit operations.

Sympathectomy tends to give excellent results at first, though the later results are not so good. However, some improvement can usually be maintained. The modern operation is a high lumbar ganglionectomy removal (L1–L2) through the lumbar route.

Before a sympathectomy is performed the behaviour of the colon filled with barium after the induction of spinal anæsthesia is noted. If peristalsis is greatly increased the patient will benefit by sympathectomy.

Presacral neurectomy with periarterial sympathectomy of the inferior mesenteric artery has also been used.

COLITIS

Classification of Inflammatory Diseases of the Colon.—

1. CATARRHAL COLITIS.
2. MUCOMEMBRANOUS COLITIS.
3. ULCERATIVE COLITIS.—
   - *a*. SIMPLE.—Follicular, stercoral.
   - *b*. NECROTIC.—Embolic, thrombotic.
   - *c*. ACUTE INFECTIVE —Typhoid, dysentery.
   - *d*. CHRONIC INFECTIVE.—Tubercle, syphilis.
   - *e*. TOXIC.—Uræmia, mercury.
4. COLITIS POLYPOSA.
5. PERICOLITIS.—SIGMOIDITIS—DIVERTICULITIS.

Catarrhal Colitis.—Colicky pains, constipation, and the passage of mucus are the chief symptoms. It is important to note that the passage of mucus may occur in simple catarrh, and is by itself no evidence of gross organic disease

IN CATARRH OF THE SMALL INTESTINE the mucus is intimately mixed with the fæces, and free bile-pigment is present. There is a marked acid reaction.

IN CATARRH OF THE LOWER COLON the mucus is present in shreds and flakes upon the outer surface of the fæces.

Mucomembranous Colitis.—The formation and passage of excessive mucus from the colon without any structural change in its wall.

AETIOLOGY.—

SEX.—Women in over 80 per cent.

AGE —Between 20 and 40 in over 60 per cent.

NEUROSIS.—In almost all the patients there is well-marked neurasthenia, and any emotional disturbance is likely to be followed by an exacerbation of the disease

GOUT and the allied conditions of lithiasis and arthritis are often associated with it. About 10 per cent pass intestinal sand.

SYMPTOMS.—

MUCUS is passed generally without mixture with fæces, in the form of long shreds or tubes. These tubes are not really membranous, but simply mucous casts of the colon in which a few cells may be entangled.

PAIN is well marked and is paroxysmal in character, chiefly in the left iliac fossa. Attacks of colic precede and accompany the passage of mucus.

CONSTIPATION is obstinate and constant, even when the colic and passage of mucus are present.

SYMPTOMS of ULCERATION, viz., passage of blood and pus, supervene in some cases. Leads to rapid emaciation.

Mucomembranous Colitis, *continued.*

TREATMENT.—

GENERAL treatment appropriate to neurasthenia.

DIET.—Coarse food, e.g., brown bread, porridge, vegetables, and fruit, to overcome the constipation.

APERIENTS usually increase the pain without curing the constipation.

LAVAGE.—Systematic irrigation of the colon with large quantities of hot water constitutes the Plombières system. It is satisfactory for a time, but relapses are usual.

SURGICAL TREATMENT (*see* p. 455).

Ulcerative Colitis.—

CAUSES —

DYSENTERY —In the tropical form, a protozoan amœba. In the epidemic or asylum dysentery, Shiga's bacillus

CATARRHAL, FOLLICULAR, AND STERCORAL ULCERS arise in any condition of constipation or colitis

PATHOLOGY.—

THE SIMPLE ULCERS occur usually in the pelvic colon, or at the flexures, or in the cæcum. They are solitary, and destroy the mucous and muscular coats, without producing much thickening of the peritoneum. Therefore they are liable to cause acute perforation Rarely their healing leads to stenosis.

THE INFECTIVE OR DYSENTERIC ULCERS may affect the whole colon. More commonly they are localized: (1) In the cæcum and ascending colon; or (2) In the pelvic colon and rectum Large areas of mucous membrane are destroyed, leaving ragged irregular ulcers In the chronic forms the gut becomes rigid and thickened and matted in adhesions.

SYMPTOMS.—

IN SIMPLE OR SOLITARY ULCERS the symptoms are merely those of catarrhal colitis Perforation is apt to occur suddenly, with almost invariably fatal consequences

IN THE MORE DIFFUSE FORMS OF ULCERATION the following occur:—

Diarrhœa is severe and persistent, especially when the rectum and pelvic colon are affected

Pain with tenesmus is similarly more severe when the lower bowel is affected.

Mucus, blood, and pus constitute the greater part of the loose stools. The association of pus with blood and mucus is the proof of active ulceration.

Tenderness along the course of the colon is specially indicative of peritoneal involvement.

Loss of flesh may become extreme. It is caused chiefly by toxic absorption, and is an important indication for surgical treatment.

MEDICAL TREATMENT.—Consists in rest, warmth, milk diet, rectal lavage, vaccines and sera, and transfusions, but this only cures a minority of the acute cases. The chronic cases are extremely resistant to treatment, and suffer from constant relapses. Polyvalent antidysenteric serum has been used with success. Sulphaguanidine is also being used successfully.

SURGICAL TREATMENT.—

Colostomy has given good results, which are due to the free drainage which it brings about. A right inguinal colostomy is probably the most efficacious, because it diverts the fæces from the colon below. The artificial anus is kept open for six to twelve months, and then closed by a plastic operation.

Appendicostomy is suitable for the less severe type of case. If it fails, one of the more radical operations can be performed later. The appendix is brought through a small parietal wound and removed, all but the proximal ½ inch This is fixed open in the wound, and through it the whole colon is irrigated daily. The circular muscle at the base of the appendix serves as a sphincter which prevents fæcal discharge. The patient soon learns to carry out the lavage at home. The appendicostomy can be closed (generally in one year's time) by the application of the actual cautery to the mucous membrane.

Ileosigmoidostomy is suitable only for cases in which the pelvic colon and rectum are healthy, and in which appendicostomy or colostomy has failed to effect a cure.

Colitis Polyposa (Multiple Polypi of the Colon).—Multiple polypi may occur in any part of the large intestine They frequently extend from the cæcum to the rectum, numbering many thousands.

The common age is between 20 and 40, and they give rise to diarrhœa and the passage of blood-stained mucus There is pain and loss of weight.

X-ray examination of the colon after a barium enema will demonstrate the polypi.

TREATMENT.—Resection of the affected portion is the only satisfactory treatment, as one or more of the polypi eventually undergo malignant change.

PERICOLITIS : DIVERTICULA : DIVERTICULITIS : AFFECTIONS OF APPENDICES EPIPLOICÆ

Diverticula may occur in any part of the alimentary tract, and may be congenital or acquired. The congenital diverticula are found principally in the duodenum and jejunum or as a Meckel's diverticulum. The acquired diverticula are limited to the colon, and the inflammatory condition associated with these is named diverticulitis

Diverticula of the Colon : Diverticulitis.—

ANATOMY.—

Situation.—Comparatively common in the descending and pelvic colon. Rare in rest of the colon Always cease at the rectum. Bulge out from the outer and inner borders of the gut, often projecting into the appendices epiploicæ (*Fig. 171*).

Number and Size—Usually multiple, and may be as many as 400. Commonly about the size of currants, they may vary from a pin's head to a grape.

Structure—Consist in a hernial protrusion of mucous membrane through the thinned muscular wall of the gut. This is specially liable to occur where a blood-vessel pierces the wall to enter a fat appendix. They often contain fæcal material, which may be inspissated to form a concretion. Rarely they lodge a foreign body.

Diverticulitis, *continued.*

AETIOLOGY.—Diverticula are caused either by increased pressure within the gut ('pulsion' diverticula), or by the dragging of a structure adherent to the outer coats of the gut ('traction' diverticula).

SEX AND AGE.—Men are more liable than women. They never occur before 20. Usually the age is about 40–60.

OBESITY is usually present, and there is much fat in the abdomen and appendices epiploicæ.

CONSTIPATION is the most important factor in their development. They indicate that muscular hypertrophy of the colon is yielding to dilatation. They are thus similar to the saccules which occur in the bladder of an old man with a large prostate.

SECONDARY PATHOLOGICAL CHANGES.—Once formed, a colic diverticulum forms a miniature appendix vermiformis, and is liable to the same pathological changes.

GENERAL PERITONITIS from perforation of, or a transudation of bacteria through, a diverticulum. The pouch may be in a condition of GANGRENOUS DIVERTICULITIS.

LOCAL ABSCESS in the left iliac fossa, exactly resembling an appendicitis abscess

CHRONIC INFLAMMATION.—A proliferative chronic inflammation occurs in the walls of the colon round the diverticulum, in the end this condition converts the gut into a thick, rigid, stenosed tube, exactly resembling some forms of scirrhous cancer.

ADHESIONS TO OTHER VISCERA.—Especially the small intestine and bladder. In the former case acute obstruction, and in the latter a vesico-colic (*Fig* 171), with the passage of flatus per urethram (pneumaturia), may result.

CARCINOMA may supervene upon the condition of chronic inflammation.

SYMPTOMS.—

LATENCY.—Diverticula often occur without causing any symptoms at all.

CLINICAL GROUPS.—The following clinical groups may be recognized:—

1. *Inflammatory.*—In this group the patient suffers from recurring attacks of greater or less severity, in which localized pain, tenderness, rigidity, and swelling are present. Temporary intestinal difficulty accompanied by vomiting may be observed. The symptoms are so similar to those caused by inflammation of the appendix that 'left-sided appendicitis' is spoken of in many instances. In the graver form an acute general peritonitis is found, and at operation a perforated diverticulum is disclosed.
2. *Obstructive.*—Chronic intestinal difficulty, with periodic attacks of exaggerated difficulty amounting to temporary obstruction, is not infrequent; in the severer cases a complete intestinal obstruction may be present.
3. *Fistulous.*—The passage of fæcal matter and air by the urethra indicates that a communication exists between the intestine and bladder (*Fig.* 171). The opening may be seen with a cystoscope. The cause is almost invariably a diverticulitis of the sigmoid flexure.
4. *Pelvic.*—An inflammatory mass is found in the pelvis, and in the female is attributed to disease of the adnexa. The symptoms are often similar to those of salpingitis and pyosalpinx.

DIFFERENTIAL DIAGNOSIS.—In cases where a tumour has formed there may be the greatest difficulty in distinguishing between carcinoma and diverticulitis. In carcinoma hæmorrhage occurs more frequently than in diverticulitis, and the discharge of mucus is more abundant. The duration of the intestinal difficulty in cases of diverticulitis may extend over years, the disease often progressing very slowly. Pelvic conditions in the female may closely resemble diverticulitis, but an X-ray examination makes the diagnosis obvious.

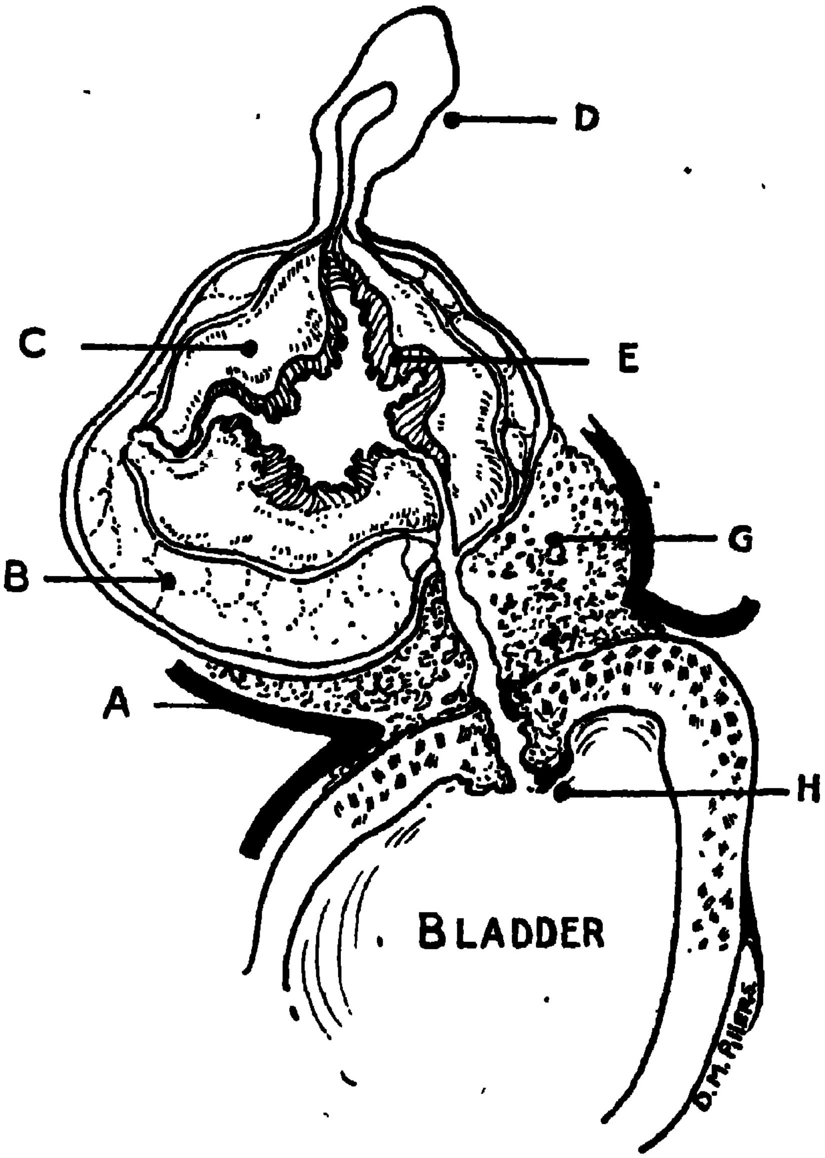


Fig. 171.—Diagrammatic representation of diverticulitis. Above is the colon and below the bladder, both in section. A, Thickened peritoneum; B, Thickened indurated wall of colon, resembling scirrhous cancer; C, Thickening of the submucous layer; D, Diverticulum extending outwards into an appendix epiploica; E, Mucous membrane; G, Inflammatory material between colon and bladder; H, Perforation into bladder.

Diverticulitis, *continued*.

TREATMENT.—

MEDICINAL TREATMENT.—The restriction of a diet having heavy residue and the administration of aperients causing fluid actions daily may check the progress of the disease and prevent its complications. In advanced cases where a tumour has formed, stenosis occurred, or fistulæ developed, surgical treatment will be necessary. The difficulty of surgical treatment, in certain cases, may lie in the extensive character of the disease, the colon from the hepatic flexure to the rectum being involved.

SURGICAL TREATMENT —Very difficult, owing to the fact that the lesions are extensive.

1 *Acute.*—
  - *a*. Excise and bury, as for appendix.
  - *b* If diverticulum cannot be found, bring colon out of wound and drain until obstruction subsides Then excise diverticulum or do a lateral anastomosis Always get large gut well washed out.
  - *c*. Wrap omentum around colon, failing other methods.
  - *d*. Excision of the affected region
2. *Chronic.*—
  - *a* Drainage above, then excise after.
  - *b*. Short circuit, if (*a*) not possible, by putting transverse colon into sigmoid colon below disease.
  - *c*. Colostomy.
  - *d* Resection is usually not possible

Chronic Sigmoiditis is a localized thickening of the pelvic colon associated with stenosis.

CAUSES —Diverticulitis (*see above*) Cicatrization of a simple or stercoral ulcer.

SYMPTOMS.—Chronic intestinal obstruction of slow onset, associated usually with a sausage-shaped tumour in the left inguinal region.

TREATMENT.—In the early stages, a milk diet with copious oil enemata may relieve Later, colostomy or excision of the affected part.

Morbid Conditions associated with the Appendices Epiploicæ.—

1. ASSOCIATION WITH DIVERTICULA (*see above*)

2 TORSION.—This results in the pedicle becoming twisted and narrow. Symptoms of recurrent colic

SEPARATION of the appendix may result, thus producing a fatty tumour free in the peritoneal cavity

INFLAMMATION may occur, with symptoms of subacute peritonitis.

3. INTESTINAL OBSTRUCTION may be caused by an inflamed and adherent appendix epiploica forming a band which strangulates a loop of small intestine

4. STRANGULATION IN HERNIAL SACS.—Long pendulous appendices may be part of or the sole contents of a hernia. Strangulation of the hernia may cause gangrene of the fatty processes. It may happen in femoral or inguinal herniæ in either sex or on either side, but the left side, femoral herniæ, and the female sex are the commonest conditions.

TUBERCULOSIS OF THE COLON

Apart from miliary tubercle, the large intestine is liable to two forms of tuberculous disease, both of which specially affect the cæcum and ileocæcal valve.

Varieties.—

ENTERO-PERITONEAL.—Both the serous, mucous, and submucous coats are attacked, with resulting caseation and ulceration. There is no excess of fibrous tissue formation, and therefore no tendency to stricture. The ileum is often involved, and also the appendix. Peritoneal adhesions, local peritonitis, and fæcal fistulæ are common.

HYPERPLASTIC.—The whole wall of the gut is involved in a dense fibrous tissue mass which surrounds the scanty tuberculous foci. Often confined to the ileocæcal valve or cæcum Produces stenosis and obstruction.

FIBRO-ADIPOSE mass surrounds the affected gut.

LYMPH-GLANDS are enlarged or involved in tuberculous disease. Those in front and behind the cæcum and those lying on ileocolic vessels are chiefly affected.

GUT WALL is transformed into a hard, rigid, fibrous mass.

MUCOUS MEMBRANE is ulcerated, or forms irregular polypoid granulations.

ILEUM AND APPENDIX are never involved in this variety.

MICROSCOPICALLY, the tuberculous giant-cell systems and bacilli are very scanty. Bacilli are seldom found in the fæces.

Age and Sex.—

THE ENTERO-PERITONEAL FORM is found at any age, with no special tendency to occur at one period of life.

THE HYPERPLASTIC FORM occurs usually between 20 and 40. Cases in children and old people are rare

The sexes are affected alike.

Symptoms.—

IN THE ENTERO-PERITONEAL FORM.—

ONSET resembles subacute appendicitis, a tender inflammatory mass forming in the right iliac fossa.

DIARRHŒA, with passage of blood and mucus.

THE INDURATED MASS becomes larger and harder, and after weeks or months a stercoral abscess bursts, and fistulæ result in the inguinal, femoral, lumbar, or umbilical regions.

LUNG COMPLICATIONS are common.

IN THE HYPERPLASTIC FORM.—

ONSET is very insidious.

CHRONIC OBSTRUCTION, with colic, gurgling, constipation, and visible peristalsis

TUMOUR forms in right inguinal region It is hard, movable, and has the form of the cæcum.

DEATH within two to three years from intestinal obstruction.

Diagnosis.—

ENTERO-PERITONEAL FORM from:—

APPENDICITIS.—In this the onset is more acute, the course more rapid. Bloody diarrhœa is rare.

ACTINOMYCOSIS.—In this the mass is harder, the parietes are more involved. Sulphur-like granules are discharged.

Tuberculosis of the Colon—Diagnosis, ***continued.***

HYPERPLASTIC FORM from:—

Cancer of the Cæcum or Colon.—This diagnosis is often not made until the mass has been removed and microscoped. In cancer the onset of obstruction and the course of the disease are more rapid. The tumour is more irregular.

Treatment.—

IN THE ENTERO-PERITONEAL FORM.—

Locally Abscesses are Opened, and usually result in fæcal fistulæ. Removal of diseased focus is impossible because of adhesive matting.

Short-circuiting the intestine above and below the disease through an opening in the midline will usually bring about a cure. Union of the ileum to the upper pelvic colon, with complete division of the former, is the most useful operation.

IN THE HYPERPLASTIC FORM.—

Excision of the affected part, together with the neighbouring lymph-glands.

Preliminary Ileosigmoidostomy may be done in cases with much distension.

REGIONAL ILEITIS

(Crohn's Disease)

Localized affection of granulomatous nature involving lower ileum. Causes cicatrization and ulceration of the mucosa.

Age.—Before 35 years. Resembles ileocæcal tuberculosis except that ileum is affected, not cæcum.

Pathology.—Lymphadenoid hyperplasia with formation of non-caseating giant-cell systems in the mucosa. Small localized abscesses may be found.

First Stage is that of peritoneal irritation—resembles subacute appendicitis.

Second Stage.—Mucosal ulceration predominates, giving symptoms of colitis.

Third Stage —Subacute or chronic intestinal obstruction.

Fourth Stage.—Adhesions and fistula formation occur.

Clinically occurs as an acute appendicitis or in a chronic stage suggestive of chronic obstruction.

X-ray Diagnosis.—In later stages terminal ileum is so constricted that it gives rise to the 'string sign'.

Treatment.—Resection of the affected segment of bowel offers the best chance of cure.

CARCINOMA OF THE COLON

Age.—Commonest between 40 and 50. Cases of cancer of the colon in patients under 30 are not so rare as in other situations.

Situation.—Of cancer of all parts of the intestine the following is the comparative incidence: rectum, 52 per cent; colon (including cæcum, ileocæcal valve, and appendix), 43 per cent; small intestine, 5 per cent.

Of cancer of the cæcum and colon the figures are: pelvic colon, 55 per cent; splenic flexure, 15 per cent; cæcum, 9 per cent; transverse colon, 8 per cent; descending colon, 4 per cent; ascending colon, 4 per cent; hepatic flexure, 2 per cent. (Burgess.)

Pathology.—

HISTOLOGY.—*Always columnar-celled growths.*

Marked hypertrophy of the muscular coats occurs both at and above the growth.

The muscular layers become invaded by columns of epithelial cells.

Small-round-cell proliferation is well marked, and becomes transformed later into connective tissue.

Colloid degeneration of both primary and secondary growths is common.

MACROSCOPIC APPEARANCE.—It is convenient to distinguish four different types of growth.

1. ANNULAR.—The growth forms a tightly constricting ring of dense hardness. The lumen is almost obliterated (*see Fig.* 165, p. 431). It is of slow development, it produces symptoms of obstruction early, and is the most favourable type for radical treatment.
2. TUBULAR.—A considerable length of the gut becomes transformed into dense new growth. Adhesions and fixation are well marked.
3. ULCERS.—The growth forms a deep, hard ulcer, with hard, fungating margins. Passage of blood and mucus results rather than intestinal obstruction.
4. FUNGATING MASS.—A soft, friable, rapidly-growing mass spreads into and along the lumen of the bowel. It causes early metastasis and free hæmorrhage, with rapid death.

THE COLON ABOVE THE GROWTH presents various degrees of hypertrophy and dilatation in proportion to the chronicity of the growth.

ULCERATION just above the growth and in the cæcum is common, and may lead to perforation.

ACUTE DILATATION WITH RUPTURE OF THE CÆCUM may occur comparatively suddenly if the growth becomes suddenly occluded, or if purgatives should have been injudiciously administered.

PERITONITIS may occur in the following ways:—

PERFORATION of a stercoral ulcer.

ACUTE RUPTURE of the dilated gut.

LOCAL SUPPURATION, leading to the formation of an abscess which may be stercoral.

PLASTIC PERITONITIS, either simple or malignant, causing a matting together of the colon with adjacent parts.

FÆCAL FISTULÆ.—

CUTANEOUS FISTULÆ are most common over the cæcum and at the umbilicus.

BIMUCOUS FISTULÆ may form between the colon and the small intestine and bladder.

INCIDENCE.—Fistula formation is much rarer in carcinoma than in the disease which may closely simulate it, viz., diverticulitis.

SECONDARY GROWTHS.—Metastatic growths are not so frequent or so early as with cancer elsewhere.

THE LYMPHATIC GLANDS which lie on the surface of the colon, and those accompanying the vessels of the part, are affected first. This form of metastasis is commonest with the soft fungating types of growth.

THE LIVER is the seat of secondary growths, especially in the late stages of the slow-growing forms of cancer.

Carcinoma of the Colon—Pathology, ***continued.***

The Peritoneum and omentum may be diffusely involved. This occurs very early in cases of colloid cancer. Extreme ascites results from this peritoneal invasion.

The Lungs and other viscera are rarely affected.

Symptoms.—

ONSET.—The onset of symptoms is usually slow and insidious, and apart from intestinal obstruction the growth may remain latent for one or two years.

Sudden Onset of acute obstruction may take place in a patient who has made no complaint of earlier symptoms. This is due to sudden blocking of a stenosed portion of the gut

GENERAL CONDITION remains good so long as the growth is confined to the colon and before obstruction is marked. The most rapid cachexia supervenes upon peritoneal invasion.

PAIN is of two kinds: (1) Intermittent Colic, due to the contractions of the gut above an obstruction, (2) Constant Pain, which occurs only when the growth has become fixed to parts outside the gut.

ABDOMINAL CONDITION.—This is characteristic only if and when obstruction exists

General Tympanitic Distension of varying grades. In obstruction to the cæcum the central abdominal area is chiefly affected; in that of the pelvic colon, the flanks

Visible Peristaltic Contractions—These are occasioned by the forcible contractions of hypertrophied gut above the obstruction Its presence always indicates a cause of long standing, and it serves to distinguish a chronic case with acute onset from one which is of recent origin The hypertrophied colon usually forms long coils obliquely disposed from above downwards, the pelvic and transverse parts being most often seen. The small intestine forms shorter transverse coils. Audible gurgling often accompanies the peristaltic contractions

TUMOUR is found in only about 40 per cent of the cases When it occurs in the pelvic colon or the splenic flexure it is specially liable to be hidden. It is movable in its early stages.

Apparent Size is increased by a fæcal accumulation above, by a matting together of neighbouring parts, or by an extension to the omentum. This latter point applies specially to cancer of the transverse colon, in which the great omentum often forms a transverse hard roll.

SPECIAL TESTS.—

Barium Mucilage per Rectum may be seen to be arrested at the site of the growth.

Barium Mucilage by Mouth is arrested above the growth.

These last two tests may be used alternately in a doubtful case

The Sigmoidoscope will demonstrate growths in the lower part of the pelvic colon.

Rectal or Vaginal Examination will often discover a tumour of the pelvic colon which is out of reach of external examination.

ACTION OF THE BOWELS.—

Constipation, though frequent, is not as pronounced as might be expected. It is often quite absent. Constipation occurring for the first time in a patient of middle life, and getting steadily worse, is suggestive of cancer of the large intestine.

DIARRHŒA alternating with constipation is the most characteristic symptom. It is due to catarrh and ulceration at or above the growth.

TENESMUS is a painful, bearing-down sensation which accompanies and follows defæcation It is present only when the growth affects the pelvic colon or rectum.

THE FÆCES may contain (1) blood, (2) mucus, (3) pus, or rarely (4) pieces of growth. They are in these circumstances peculiarly offensive, and indicate a rapid ulceration or necrosis of the growth.

Complications.—

ACUTE INTESTINAL OBSTRUCTION (*see* Chapter XXXVII), which may be first indication of disease, is brought on by. (1) Obstruction of the lumen of the bowel by a fæcal mass; (2) Adhesions or kinking; (3) Paralysis due to abuse of purgatives or morphia.

ACUTE PERFORATION usually results from the rupture of a stercoral ulcer. The resulting peritonitis is almost invariably fatal, and is often associated with a subnormal temperature.

FISTULÆ —AN EXTERNAL FISTULA may actually relieve the distension. AN ENTEROCOLIC FISTULA will cause persistent diarrhœa and rapid marasmus. A GASTROCOLIC FISTULA causes lientery, in which undigested food is passed in the fæces, and true fæcal vomiting. A VESICOCOLIC FISTULA causes cystitis, with the passage of gas and fæces per urethram. A VAGINOCOLIC FISTULA may relieve the obstruction by the passage of gas and fæces by the vagina.

METASTASIS —ASCITES from multiple peritoneal growths is the commonest sign. THE LIVER may become enlarged and nodular, but secondary growths often occur without this sign.

TOXÆMIA is late in development, and is, next to obstruction, the commonest cause of death.

Course of the Disease.—Uncomplicated cases last from two to five years. The hard and constricting growths have the best prognosis, because of their slow growth, scanty metastasis, and also because they are quickly recognized by the resulting chronic obstruction.

AFTER COLOSTOMY patients often live for a year or more.

Treatment.—Only a minority of cases permit of radical surgical treatment. In the remainder, apart from palliative operations, the chief treatment consists in a light diet of food which contains but little indigestible debris. Milk, soups, and light farinaceous food form the staple, and fish, eggs, and a little meat are allowed. Vegetables, brown bread, and fruit are specially harmful.

EXPLORATORY OPERATION should always be advised in cases of doubtful diagnosis. Especially when the following occur: (1) Constipation of recent origin and progressive degree; (2) Passage of blood, mucus, and pus by rectum; (3) The existence of visible peristalsis.

SITE.—The midline below the umbilicus gives access to all parts except the splenic flexure. This may have to be explored through a second incision in the upper part of the left semilunar line.

COLOSTOMY may be done as a palliative or preliminary operation.

PALLIATIVE.—Must be above the growth. Either in the pelvic colon, the transverse colon, or the cæcum, the first being much the most

Carcinoma of the Colon—Treatment, *continued*.

frequent. An *axial colostomy* is the best in these cases: the bowel is divided, and a large tube tied in the proximal and a small tube in the distal end.

If the bowel cannot be brought outside the abdomen, a *lateral colostomy* must be done.

If there is no urgent obstruction, the operation ought always to be done IN TWO STAGES, the bowel being brought outside the abdomen and fixed at the first stage, and opened or divided at the second.

PRELIMINARY COLOSTOMY.—If there is a chance of radical removal of the growth, this ought always to be preceded by a colostomy or short-circuiting operation. It is of great advantage to do this at some distance from the growth, because it allows the radical operation to be done in a clean area, e.g., a transverse colostomy should precede excision of a pelvic growth, or a cæcostomy one of the transverse colon.

ANASTOMOSIS OPERATIONS.—These have the same indications as colostomy, but they are possible only in the absence of marked obstruction. A short-circuiting operation has these great advantages over a colostomy: (1) When performed as a palliative operation it avoids the unpleasantness of an artificial anus; (2) When done as a preliminary to excision, the latter operation is simplified to a mere removal without anastomosis. The best short-circuiting operation is an ileo-sigmoidostomy. The ileum should be divided after a lateral junction with the pelvic colon, unless the growth is inoperable and likely to cause complete occlusion, when the lumen of the ileum should be preserved.

RESECTION OF THE GROWTH.—

CONTRA-INDICATIONS.—Metastasis in the liver or peritoneum. Extensive adhesions, or great lymphatic involvement.

EXTENT OF RESECTION.—A long piece of gut should be removed, including several inches above and below the growth, together with a fan-shaped area of peritoneum having its apex at the root of the mesentery. This will include all the lymphatic vessels and glands connected with the growth. Sulphaguanidine or succinyl sulphathiazole is given pre- and post-operatively to reduce the intestinal flora.

ADVANTAGES OF SECONDARY RESECTION.—In all cases the removal of the growth should be preceded by a colostomy or short-circuiting operation (e.g., ileosigmoidostomy). The advantages of this are:—

1. The intestinal obstruction and toxæmia are relieved before the excision.
2. The chances of sepsis are diminished.
3. Shock is greatly reduced.
4. The malignant mass often becomes smaller and less adherent owing to the relief of congestion.
5. A microscopical examination of the mass can be made before excision.
6. The mortality of the operation is reduced from about 50 per cent to 10 per cent.

CHAPTER XL

DISEASES OF THE RECTUM AND ANUS

CONGENITAL MALFORMATIONS OF THE RECTUM

1. ABSENCE OF RECTUM AND ANUS —Colon ends at the pelvic brim. The pelvis is narrow and ill-developed. No bulging occurs in perineum when child cries.
2. RECTUM IS PRESENT, BUT THE ANUS IS ABSENT.—Bulging occurs in the perineum on crying.
3. ANUS AND ANAL CANAL ARE PRESENT, BUT DO NOT JOIN THE RECTUM.—Finger feels a partition about one inch above anus.
4. A STRICTURE MAY EXIST where anal canal joins rectum.
   TREATMENT OF FOREGOING CONDITIONS.—
   - *a.* Dilate a stricture by daily passage of bougies.
   - *b* Perforate a septal division, and then dilate.
   - *c.* Where anus is imperforate · Cut upwards in midline for two inches; bring rectum down, open, and sew to skin.
   - *d.* If the rectum cannot be reached from the perineum: Perform inguinal colostomy.
5. RECTUM OPENS INTO OR COMMUNICATES WITH BLADDER, VAGINA, URETHRA, OR VULVA.—
   TREATMENT.—By a plastic operation in slight cases in later life, after preliminary colostomy to divert the fæces.

PROCTITIS, OR INFLAMMATION OF THE RECTUM

Causes.—Foreign bodies—Fish-bones or other hard material in fæces—Polypus or tumour of the rectum—Piles, fissure, or fistula—Prolapse—Dysentery — Syphilis — Tuberculous ulceration—Gonorrhœa—Parasites: threadworms (in children), bilharzia (in patients from Africa)—Lymphogranuloma inguinale.

Symptoms.—Bearing-down pain. Painful tenesmus. Feeling of weight and fullness in perineum. Discharge of muco-pus from the anus.

Treatment.—

1. Ascertain and remove the cause, e.g., piles, fissure, or foreign body.
2. Copious hot-water enemata. Lead and opium lotion or suppositories in acute stage. Lotio protargol, gr. j ad ℥j, in later stages. Tannic acid, 15 per cent, as a wash-out. Gentian violet capsules by mouth.
3. For threadworms: Purge, and inject with infusum quassiæ.
4. For bilharzia: Scrape away the adenomatous polypi which contain the eggs.
5. Incision of sphincter or colostomy if other means fail.

ISCHIORECTAL ABSCESS

Anatomy.—Ischiorectal fossa is triangular in vertical section. Outer wall is formed by ischium clothed by obturator internus. Inner wall is formed by the rectum clothed by levator ani. Lower wall is formed by the skin. In front is the triangular ligament. Behind is the border of the gluteus maximus.

Sources of Infection may be: Through any of the three walls or from in front.

1. Through rectum by ulceration or perforation of foreign body—the common route.
2. Through skin by wound abrasion or sebaceous gland
3. Through the outer wall from disease of pelvis
4. From the urethra in front.

Varieties.—

1. CELLULITIS.—Possibly gangrenous. Diffuse cellular infection from the bowel or skin Generally in feeble old people.
2. ACUTE ISCHIORECTAL ABSCESS —*Bacillus coli* infection from the bowel, or a pyogenic infection from the perineum.
3. CHRONIC ABSCESS.—Generally tuberculous in origin.

Symptoms.—Those of local inflammation Brawny swelling in the perineum. Hot, very painful, swelling felt per rectum. Defæcation is very painful Bursts either through the skin or into the bowel, or in both directions at once. Often results in a fistula

Treatment.—

ACUTE CASES.—Open freely from the perineum, and drain. Drainage must be early and free and before the classical sign of suppuration is detectable. If an opening exists into the bowel, treat like a fistula.

CHRONIC CASES.—Try scraping and packing with sulphanilamide and penicillin powder. Liability to sepsis makes healing bad. Colostomy has often to be performed

FISTULA IN ANO

Definition.—An abnormal communication between the rectum or anal canal and the exterior (N B —The blind external fistula and blind internal fistula are thus not true fistulæ at all, but in their natural course are likely to become so.)

Varieties (*Figs.* 172, 173, 174).

1. COMPLETE —Track opens into the rectum internally, on to the perineum externally.
2. INCOMPLETE EXTERNAL —A perineal sinus not opening into the rectum.
3. INCOMPLETE INTERNAL.—A track opening into the rectum, but not externally

Causes.—The causes of proctitis. Ischiorectal abscess. Ulceration or stricture low down in the rectum: malignant, tuberculous, or syphilitic.

Anatomy.—A suppurating or granulating sinus communicates with one or both openings. It runs outside the bowel entirely, or more frequently perforates the gut obliquely, so running some distance between mucous membrane and muscular coat. May almost encircle the bowel. The internal opening is usually single, however many external openings there are. The external openings are usually multiple. The track runs either through the fibres of the external sphincter or, more commonly, superficial to it. The fistula, like the abscess from which it originates, is chiefly: (*a*) Ischio-rectal; (*b*) Pelvic; or (*c*) Submucous.

IN CHRONIC CASES: Dense cicatricial tissue forms round the sinus. Multiple diverticula branch off in various directions, especially upwards between the sacrum and anus. Both internal and external openings become multiple.

Symptoms.—Pain, especially at defæcation. Purulent discharge from a sinus, or from anus with or after the motion.

Treatment.—

PRELIMINARY.—Enema just before operation.

OPERATION.—Pass a director up the fistula. Open it completely from end to end. Scrape out all granulations and cut out scar tissue. Open up all secondary channels and sinuses. Pack deeply with oiled gauze.

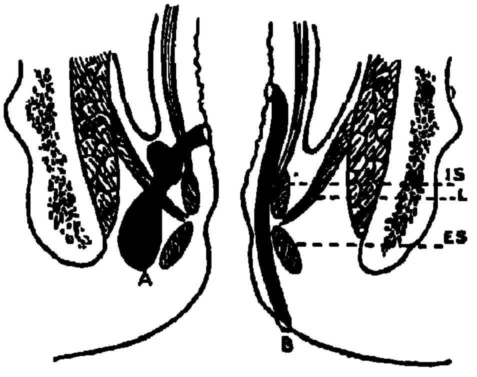


Fig. 172.—Diagram of fistulæ in ano. I.S., Internal sphincter; E.S., External sphincter; L.A., Levator ani; A, Blind internal fistula, partly pelvic and partly ischiorectal, B, Complete submucous fistula.

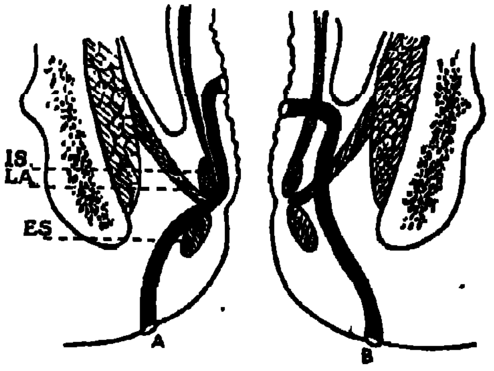


Fig. 173.—Diagram of fistulæ in ano. I.S., Internal sphincter; E.S., External sphincter; L.A., Levator ani, A, Complete submucous and ischiorectal fistula; B, Complete pelvic and ischiorectal fistula. The dangerous variety.

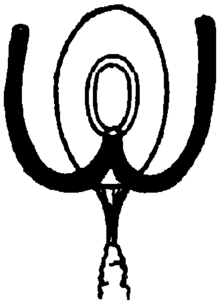


Fig. 174.—Diagram of horseshoe fistula of the anus. There is a single central internal opening and a double lateral external opening; the fistula runs nearly all round the anal canal. (*After Miles.*)

Fistula in Ano—Treatment, ***continued.***

IN DEEP FISTULÆ WHICH ARE PELVIC IN ORIGIN.—If the fistula opens above the internal sphincter, it is dangerous to divide that muscle and the levator ani, because fæcal incontinence will result. The fistula must be opened freely from the perineum by a T-shaped incision and allowed to granulate from the bottom. Alternatively, the operation of excision may be performed in two stages.

IN CHRONIC OR COMPLICATED CASES.—Either perform a perineal excision of the rectum, or an inguinal colostomy.

FISSURE IN ANO

Definition.—A longitudinal ulcer in the anal margin.

Situation, etc.—Usually single and in mid-posterior line, where the anus is most firmly fixed to the coccyx.

Below the fissure is a 'SENTINEL PILE', which is a tag of mucous membrane that has been torn down by a scybalous mass, so forming the ulcer.

Rather commoner in men than women.

Symptoms.—Severe burning pain during and after defæcation, with great tenesmus. Fæces may be streaked with blood.

Treatment.—

PALLIATIVE.—Laxatives and copious hot enemata. Percaine suppositories and the unguentum gallæ c opio.

INJECTION.—In mild cases or recent cases healing can be secured by injecting the external sphincter with A B.A (a local anæsthetic with prolonged action), thus securing rest for the ulcer for two or three days. Useless in chronic cases.

OPERATIVE.—Under an anæsthetic: (1) Dilate the sphincter; (2) Remove all piles, especially 'the sentinel'; (3) Cut out the ulcer, and divide the pecten band.

FIBROUS STRICTURE OF THE RECTUM

Aetiology.—Elderly patients. Usually women. Follows: (1) Ulceration due to dysentery, tubercle, or syphilis; (2) Cellulitis contracting outside the gut; (3) Other conditions of proctitis, e g., that due to gonorrhœa or piles.

Anatomy.—Stricture is situated two or three inches from the anus. It may be: (1) Annular and smooth; (2) Long and irregular; (3) Tied to the sacrum. The gut above is hypertrophied, dilated, and ulcerated. The gut below is often ballooned.

Symptoms.—Alternating attacks of constipation and diarrhœa. Attenuated moulded fæces, with scanty blood and mucus. Chronic intestinal obstruction. Ischiorectal or pelvic abscesses may form by rupture or infection through a stercoral ulcer. Large fæcal abscesses may form in the buttock, and cause death or chronic fistulæ.

Treatment.—

DILATATION by graduated bougies, when these can be borne.

EXCISION of the stricture when it is short and the bowel above and below healthy and free, or linear proctotomy.

INGUINAL COLOSTOMY in the worst cases.

SYPHILIS OF THE RECTUM AND ANUS

Primary Chancre.—Is rarely situated at the anus, but may occur in the anal canal.

Secondary Condylomata.—Are very common round the anus.

Tertiary Gummata.—Form a diffuse infiltration of the rectal wall. They are commoner in women than men. The disease begins in the submucous tissue. Ulceration soon follows, and is deep but comparatively painless. It may extend to the anus, sigmoid, vagina, perineum, or bladder, and form fistulæ. It usually results in an extensive stricture.

SYMPTOMS.—Some pain and rectal tenesmus. Discharge of blood and pus per anum. Later, the signs of chronic obstruction.

TREATMENT.—

1. SALVARSAN, SULPHONAMIDES, AND IODIDES until all active disease has been arrested.
2. Temporary or permanent COLOSTOMY.
3. In a very few cases when the stricture is short, EXCISION.
4. Resulting stricture to be treated by BOUGIES.

NEW GROWTHS OF THE RECTUM AND ANUS

Polypus (*or Stalked Adenoma*).—Consists of simple gland tissue with more or less connective tissue connected to the submucous coat by a vascular pedicle. It is commonest in children. It may cause some rectal pain, irritability, or prolapse.

TREATMENT.—By ligature and removal.

Papilloma.—May be localized or a diffuse growth, with a tendency to free hæmorrhage. It is of rare occurrence.

TREATMENT.—By removal.

Submucous Lipoma.—Can be completely excised.

Bilharzia Papilloma.—This is a polypoid outgrowth of mucous membrane, bleeding freely, in which many bilharzia ova are found.

Epithelioma.—Attacks the anus. Often in the site of an old fissure. It may form a warty mass, but soon becomes ulcerated. It is indurated, its edges are hard and everted. It bleeds very freely, and has a copious foul discharge. Secondary deposits occur very early in the inguinal glands.

TREATMENT.—By early free removal, together with the glands in both groins.

Deep X-ray therapy has given good results in some cases.

Sarcoma.—This is a rare disease, occurring in young patients (under thirty). It forms a rather diffuse growth in the submucous tissue which causes obstruction. Ulceration is absent or late.

TREATMENT.—It should be removed, but recurrence is likely.

Carcinoma.—*See below.*

CARCINOMA OF THE RECTUM

Aetiology.—A little commoner in men than in women, and occurs generally after middle life.

Carcinoma of the Rectum, ***continued.***

Anatomy.—It begins in the mucous membrane, and thence spreads: (1) Into the lumen of the gut; (2) Up and down the gut; (3) Out through the gut wall in neighbouring structures.

ITS MACROSCOPIC STRUCTURE may be of three types:—

1. A FUNGATING FRIABLE MASS projecting into the bowel.
2. AN ULCER with hard raised margin, of considerable extent (*see Fig.* 22, p. 67).
3. A FIBROUS ANNULAR CONSTRICTION with comparatively little new growth or ulceration

The bowel above and below shows the same changes as are described in the section on intestinal obstruction (p. 416).

MICROSCOPICALLY it is always a columnar-celled carcinoma, which often shows colloid degeneration, and in which the proportion of glandular and fibrous tissue varies. Thus, in the soft, rapidly growing kinds, the glandular tissue predominates, whilst the hard annular varieties consist chiefly of fibrous tissue.

Extension.—Occurs outwards through the bowel wall, and may involve: (1) The cellular tissue; (2) The sacrum; (3) The pelvic vessels and nerves; (4) The vagina or uterus, (5) The prostate, bladder, and ureters. In the case of the vagina or bladder a bimucous fistula may result by ulceration.

Metastasis.—This is comparatively late and rare. It occurs: (1) In the lumbar glands, (2) In the liver and peritoneum; (3) In the inguinal glands when the anus has been involved.

Symptoms and Signs.—

1. EARLY, BEFORE OBSTRUCTION —

   PAIN of a dull, dragging kind, with some tenesmus. This may be quite absent.

   BLEEDING both with and between the motions.

   Passage of offensive blood-stained mucus

   Rarely the motions may become narrow and moulded

   DIARRHŒA alternates with constipation.

   ON EXAMINATION, a mass is found which is either (*a*) Friable and fungating, or (*b*) Hard, with ulcerated surface and everted margins. The mass is not fixed in any way, and on straining the bowel descends as a whole.

2. ADVANCED, WITH OBSTRUCTION.—The signs and symptoms of chronic intestinal obstruction are added to the above. Great abdominal distension, with visible peristalsis. Pronounced constipation, with colicky pain, which is increased by purgatives. Pain and tenesmus are much more constant and severe

   ON EXAMINATION, the lumen of the bowel is partially obliterated by a stricture or fungating mass.

3. INVOLVEMENT OF NEIGHBOURING STRUCTURES.—Severe constant or neuralgic pain is caused by extension to the sacrum and sacral nerves. Œdema, by involvement of the vessels. Gas or fæces may be voided from the urethra, and cystitis supervene from bladder extension. Hydronephrosis, pyonephrosis, and uræmia, from blocking of the ureters. Gas and fæces may be passed from the vagina.

   An abscess may form in the pelvis, ischiorectal fossa, or buttocks, and give rise to one or many fistulæ, which communicate with the gut above the growth.

ON EXAMINATION, the growth is fixed and the bowel does not come down at all on straining. This fixity may be only partial, e.g., to the bone behind or vagina in front, or it may be complete, the whole bowel circumference being involved.

4. LATE CACHEXIA with secondary growths.—This stage is often never reached, the patient dying either from intestinal obstruction or sepsis. Masses of glands may be felt in lumbar regions. Liver becomes large and nodular: ascites develops.

Course.—Varies very much. In young patients and with soft fungating growths the whole course may be only one year. In older patients and with fibrous growths it may last five years.

Diagnosis.—This is usually easy if the case is examined locally. In the early stages IT IS IMPOSSIBLE to diagnose cancer from piles, prolapse, polypus, or fistula WITHOUT A RECTAL EXAMINATION AND PROCTOSCOPY.

IN PILES there is no new growth, but merely swollen veins, but it is important to remember that piles often complicate cases of cancer, so that the inside of the bowel should be examined in every case of piles.

IN FISSURE the pain is much more severe, and is almost unbearable on digital examination. A linear ulcer with sentinel pile is evident, and the inside of the bowel is healthy

IN POLYPUS the mass is small and pedunculated, and the mucous membrane healthy

IN FIBROUS STRICTURE the parts are indolent and there is no new growth. Bleeding on examination is absent or very slight.

IN SYPHILITIC DISEASE the gummata are soft, painless, and non-vascular. The ulcers are ragged, undermined, and not at all indurated.

IN PROLAPSE AND INTUSSUSCEPTION inspection and palpation will make the case clear

NOTE.—THE SIGMOIDOSCOPE is useful for cases of cancer occurring out of reach of the finger (three to twelve inches from the anus). EVERY CASE OF BLEEDING PER ANUM SHOULD BE SIGMOIDOSCOPED.

Treatment.—

CASES UNSUITED FOR EXCISION—Growth is fixed to the bladder, prostate, or ureters, or to the sacrum high up. Extension into the cellular tissue or glands Metastatic growths. Conditions of age and debility.

TREATMENT.—Soft food with little debris, avoiding fruit, vegetables, etc. Bowel washed out through a soft tube daily. Olive oil enemata.

COLOSTOMY for obstruction.

LOW OPERATION: PERINEAL EXCISION.—

INDICATIONS.—For cases in which the disease extends within one inch of the anus, but not higher than the finger can reach.

METHOD.—After a preliminary colostomy, the anus is excised with the lower end of the bowel well above the growth. The wound is either plugged round a central tube without any stitching of the gut to the skin, or a few anchoring stitches may be used.

ABDOMINO-PERINEAL METHOD.—This is the operation of choice, but owing to its severity is contra-indicated in the feeble.

INDICATIONS.—May be used for all cases; it is specially suitable for cases in which the disease extends above the 3rd sacral vertebra.

Carcinoma of the Rectum—Treatment, ***continued.***

It has the advantage of allowing a thorough exploration of the abdomen for metastatic growths, of allowing a free removal of lymph-glands and cellular tissue from the pelvis, and of making a much freer removal of bowel possible (*Fig.* 175).

If necessary a preliminary colostomy can have been performed. With the patient in the Trendelenburg position, the abdomen is opened through a median incision and explored as to operability of the growth and the presence of secondary deposits. If growth is operable, pack off small gut and divide the mesenteric artery at level of bifurcation of the aorta. Peritoneum divided on either side of the rectum and the mesorectum stripped forwards from the hollow of the sacrum. Sigmoid colon divided between clamps and the upper end brought out through a left inguinal incision as a colostomy; the lower end is pushed down into the hollow of the sacrum and the pelvic floor reconstituted by uniting the peritoneum. Abdomen is closed, patient placed into lateral position, and the bowel removed through the perineal route.

Results of Excision of Cancer of the Rectum.—

IMMEDIATE MORTALITY is about 15 per cent.

SURVIVAL.—Of those recovering from operation, 60 per cent survive for three years or longer The prognosis of survival is very bad in cases where the anus is involved. Such cases seldom if ever survive three years.

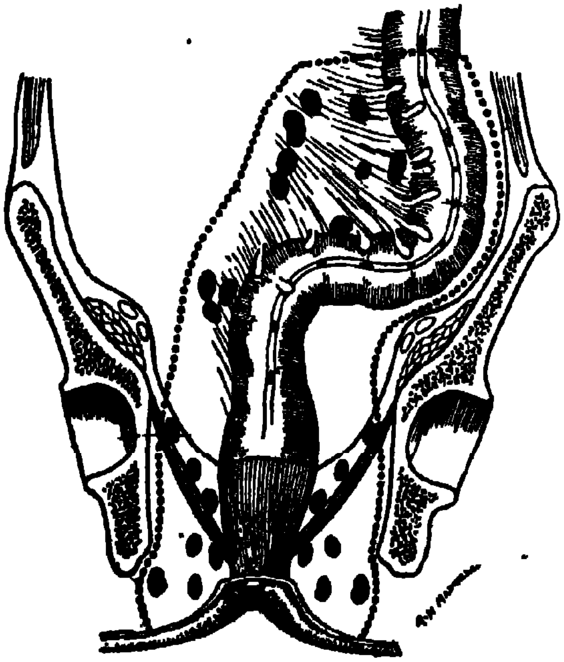


Fig. 175.—Diagram of the parts removed by abdomino-perineal excision of the rectum, indicated by the dotted line. The black spots are the lymph-glands involved by cancer of the rectum. (*After Miles.*)

RECURRENCE.—In about two-thirds of the cases when recurrence takes place it occurs within two years of the operation. The site of recurrence in 74 per cent is local in or round the rectum; in 24 per cent is in the form of visceral metastasis; and in 4 per cent in the lymphatic glands. The average length of life of those in whom recurrence occurs is about two years.

HÆMORRHOIDS OR PILES

Definition.—Varicose veins of the anal region.

Causes.—Pressure of the blood in the portal system. Absence of valves in the portal veins. Anastomosis between the portal and systemic system that occurs between the superior, middle, and inferior hæmorrhoidal veins. Any portal congestion, cirrhosis of the liver, or intra-abdominal tumour. Alcoholism and chronic constipation.

Specially common in young men and middle-aged pregnant women

Varieties.—External and internal.

External Piles.—These are covered by skin.

CONSIST of a central vein covered by a tag of hypertrophied skin.

SYMPTOMS are slight. Pruritus and some pain. Inflammation and rupture of one of the contained veins, forming a perianal hæmatoma, are frequent.

TREATMENT.—By hamamelis ointment and careful washing Fomentations when inflamed. Excision must be sparingly done, so as to avoid excessive cicatrization. Incision of the perianal hæmatoma and evacuation of the clot.

Internal Piles.—Occur inside the sphincter, and may extend two inches up the bowel.

CONSIST OF varicose veins covered by hypertrophied mucous membrane. They form a series of fleshy, purple masses inside the anus, some being thick, firm, and fleshy, and others thin-walled and easily bleeding.

SYMPTOMS.—Pain and dull aching after defæcation. Very sharp pain is due to a fissure.

BLEEDING after defæcation, the blood being spattered over the pan.

MUCOID DISCHARGE, with tendency to prolapse.

COMPLICATIONS—

INFLAMMATION and thrombosis. If suppurative, may cause portal pyæmia.

STRANGULATION and sloughing by pressure of the sphincter on prolapsed piles.

PROLAPSUS RECTI.

FISSURE IN ANO.

TREATMENT.—

GENERAL —Saline aperients, and plenty of exercise.

LOCAL.—Washing after defæcation. Astringent ointments, e.g., the unguentum hamamelidis or unguentum gallæ c. opio.

BY INJECTION.—Each pile is injected at its base with 2 c c. of 5 per cent solution of phenol in almond oil. Several treatments may be required. An anæsthetic is not necessary, nor does the patient need to lie up afterwards.

Internal Piles—Treatment, ***continued.***

OPERATIVE.—Where bleeding or pain is severe, and where no serious permanent portal congestion, e.g., cirrhosis, exists.

Preliminary.—Purgative, e.g., castor oil following salines. Discontinue these forty-eight hours before. Enema twelve hours before operation.

Lithotomy position. Piles are drawn down by forceps and clamped. Piles removed and the bases oversewn with catgut Morphine suppository (½ gr.) inserted.

Bowels are kept confined for six days after.

PROLAPSE OF THE RECTUM

INCOMPLETE.—Is commonly associated with piles in adults, when only the mucous membrane protrudes.

COMPLETE.—Is common in children, when all the coats are extruded.

Cause.—(1) Relaxed conditions of the tissues; (2) Constant straining, e.g., large prostate, (3) Diarrhœa or worms; (4) Chronic constipation, piles.

Signs, etc.—Protruding mass at the anus, with central orifice communicating with the lumen of the rectum, its walls joining the skin at the anal margin. Mucoid discharge and bleeding Incontinence of fæces when it is chronic. Strangulation and sloughing are very rare.

DIAGNOSIS from piles, polypus, and intussusception

Treatment.—

PALLIATIVE.—Removal of any cause of straining or any rectal irritation, e.g. worms or piles.

ASTRINGENT LOTIONS.—Defæcation in a lateral position the last thing at night; a pad soaked in the lotion is tied on with a T-bandage over the anus.

OPERATIVE —

Removal of the mucous membrane in incomplete prolapse.

Linear scarification, injection of astringents or of paraffin.

Excision of the whole projecting tumour, with suture of the bowel, in complete cases.

Colopexy, or suture of the sigmoid flexure to the parietes, in a very few relapsing cases

Incision into the space between the anus and coccyx. This is then packed with antiseptic gauze and the space allowed to heal by granulation from the bottom. The contraction of this holds the bowel in position.

PRURITUS ANI

Definition.—A condition characterized by intense itching around the anus.

Causes.—

1. IN ADULTS.—The common cause is a constantly damp condition of the anal skin due to leakage. This may be due to: (*a*) Internal piles or polypus, by prolapsing into the sphincter; (*b*) Fissure; (*c*) Fistula; (*d*) Catarrhal proctitis; (*e*) Fungus infections of the anal margin, monilia and epidermophyton.
2. IN CHILDREN.—Almost always due to worms.

Pathology.—The skin around the anus is moist and corrugated, and usually shows abrasions from scratching. Pruritus is only a symptom arising from some primary cause, but it may continue after the original cause has cleared up. Probably after existing some time changes take place in the skin and nerves resulting in a true sensory neurosis.

Clinical Features.—Intense itching is complained of, beginning just at the margin of the anus. The most intense irritation is generally just at the opening of the anus and along the median raphe in front and behind. Sometimes it is more or less continuous, while at other times it comes on in bouts. It is generally worse at night. Many patients are quite unable to sleep for weeks and have their lives rendered utterly miserable. They often state that the intense irritation is more difficult to bear than pain. It is much commoner in men than in women.

Treatment.—

A careful examination of the rectum and anus must be made, and any local condition removed.

Careful washing of the surrounding parts, and afterwards applying 1–40 carbolic lotion or a dusting powder, often relieves Injection of oil-soluble anæsthetics—e g., A.B.A , proctocaine. Treatment of fungus infections with brilliant green (1 per cent) or gentian violet (25 per cent).

Ball's operation, in which, by dissecting up a flap of skin around the anus, the nerves are divided, gives good results.

CHAPTER XLI

AFFECTIONS OF THE LIVER AND BILE-DUCTS

WOUNDS OF THE LIVER

Non-penetrating Wounds (Subcutaneous Rupture).—

CAUSES.—Buffer accidents—Running over—Blows, kicks, falls, etc.—Fracture of ribs.

PREDISPOSING.—Fatty or congested liver.

VARIETIES.—

CONTUSION.—Ecchymosis. Glisson's capsule is unruptured.

RUPTURE.—Glisson's capsule ruptures. A portion of liver may be detached completely.

SITUATION, ETC.—Right lobe most common. Antero-posterior laceration May be transverse in superior surface if caused by doubling up of liver.

SYMPTOMS.—

SHOCK, from violence of accident

COLLAPSE, from internal hæmorrhage.

BRUISE, from point of application of force over the liver

PAIN over liver down to umbilicus and up to scapula or ensiform cartilage.

PERITONEAL HÆMORRHAGE.—Abdomen is held rigid Shifting dullness appears in the flanks; most marked on the right side. Pulse becomes increasingly rapid The distension and dullness increase rapidly.

URINE may contain bile or sugar.

PULMONARY EMBOLISM occasionally results from liver tissues forming an embolus.

FRICTION SOUNDS over the liver may be heard later.

TREATMENT.—Usual treatment for shock. Open abdomen if hæmorrhage is evident or collapse continues. Incise through linea alba. Stitch up liver, or pack with gauze and leave in for from 5 to 7 days. Purse-string sutures may be used for small wounds. In difficult cases the rent may be plugged with a piece of great omentum. Incision may be enlarged by cutting round costal margin.

Penetrating (External) Wounds.—Stabs—Gunshot—Lacerated, etc. (impaled). Much rarer than rupture.

SYMPTOMS.—Shock and collapse—External or internal hæmorrhage (rapidly fatal)—Escape of bile—Vomiting—Hiccup.

LATER SYMPTOMS.—Tympanitic distension of abdomen—Rigors—Delirium—Peritonitis—Secondary hæmorrhage—Abscess.

DIAGNOSIS from position, direction, and depth of wound.

TREATMENT.—If hæmorrhage and shock increase, open abdomen and plug or suture. In gunshot wounds, remove missile if possible.

WOUNDS OF GALL-BLADDER OR DUCTS

Penetrating Wounds.—

CAUSES and SYMPTOMS as in wounds of liver, but escape of bile is more marked. Bile soon makes its appearance in the urine.

Rapid Development of Peritonitis.

TREATMENT.—

Complete exposure by laparotomy.

Wound of Gall-bladder.—Sew up if small, and close abdomen. Sew to abdominal incision if large, or remove gall-bladder.

Wound of Cystic Duct.—Cholecystectomy.

Wound of Hepatic Duct.—Suture ends of duct.

Wound of Common Bile-duct.—Tie open ends. Cholecystenterostomy.

Rupture of Gall-bladder or Ducts.—

CAUSES.—Traumatism: kick, blow, run-over, etc., especially when gall-bladder is distended and a calculus is present—Ulceration from calculus—Typhoid fever—Ascaris—Malignant disease (very rare)—Dilatation from blocking duct.

PATHOLOGY.—

If Sudden and Extensive: General peritonitis results rapidly.

If Gradual : An encysted fluid collection amounting to several gallons may result.

Continuity of bile-duct may be restored in few weeks in latter case, and this has been proved experimentally.

SYMPTOMS.—Either immediate septic peritonitis, or localized peritonitis. followed by fluctuating tumour—Jaundice—Bile in urine—Clay fæces.

DIAGNOSIS is that of acute peritonitis associated with traumatism, jaundice, or history of gall-stone colic.

TREATMENT.—

In Subacute or Obscure Cases.—Keep in bed. If localized swelling occur, laparotomy.

In Acute Cases.—Immediate laparotomy. Details as in case of penetrating wounds (*see above*). A septic gall-bladder should always be drained, or removed.

CHOLECYSTITIS

Definition.—Inflammation of the gall-bladder. This is usually associated with the presence of gall-stones, and the latter by their mechanical effects dominate the clinical picture, but cholecystitis may be a primary condition.

Aetiology.—Caused by infection by *B. typhosus*, *B. coli*, streptococci, or staphylococci.

Varieties.—

EARLIEST FORM.—This is merely a catarrhal inflammation, associated with a villous thickening of the mucous membrane in which cholesterol is deposited. This type is known as the *strawberry gall-bladder* or *cholesterosis*.

Symptoms.—Vague discomfort or right hypochondriac pain with vomiting and constipation.

Cholecystitis—Varieties, ***continued.***

LATER OR MORE ACUTE VARIETIES.—Produce distension of the gall-bladder with muco-pus, massive thickening, and dilatation or sloughing. A local peritonitis or abscess may occur from perforation, but general peritonitis is very rare.

SYMPTOMS.—Localized pain, with tumour formation. Vomiting, constipation, and fever.

Treatment.—Removal of the gall-bladder. In the gangrenous type, or when a local abscess has formed, the gall-bladder must be drained as a first stage, and removed later if a chronic fistula results.

GALL-STONES

Causes.—

1. STAGNATION OF BILE —From sedentary habits, corsets which impede the flow of bile by impeding respiratory movements, and constipation The bile-salts are said to undergo decomposition so that the bile becomes acid and cholesterol is precipitated. There is very little real evidence to support this view. In obstruction of the common duct from carcinoma, although the bile becomes extremely viscid, stones, cholesterol or otherwise, are never formed.
2. INFECTION OF THE GALL-BLADDER —Naunyn showed that the cholesterol comes from the epithelium of the gall-bladder and not from the bile The formation of gall-stones depends on the presence of an inflammatory catarrh of the gall-bladder mucous membrane in which the amount of cholesterol is greatly increased, the desquamated epithelial cells and bacteria acting as a nucleus.

   CAUSE OF THE INFECTION —The common organisms are *B. coli* and *B. typhosus* These have been found in the gall-stones. There is often a history of previous typhoid fever The path of the infection is probably the blood-stream. It is probable that stones are only formed if the infection is chronic and attenuated, if acute, the excess of cholesterol does not occur. The frequency of gall-stones in women is probably due to the great frequency with which *B coli* infections occur in women who have been pregnant
3. FOREIGN BODIES (rare).—Needle, ascaris, distoma, hydatid, etc

Seat of Formation.—Generally in gall-bladder. May be found in cystic or common duct—more rarely hepatic duct. (*Fig.* 176.)

Structure.—Three layers: Nucleus—Middle layer—Outer layer.

NUCLEUS —Consists of: Clump of bacteria—Mucus—Blood-clot—Casts of bile capillaries, or foreign body, or crystals of $CaCO_3$.

MIDDLE LAYER.—Laminated layers of cholesterin crystals.

EXTERNAL LAYER.—Bilirubin combined with a calcium salt.

Varieties.—Compounds of cholesterin and bilirubin-calcium. Either substance nearly pure. Latter in calculi formed in liver.

Incidence.—In 4 per cent of all males; in 20 per cent of all females; in about same proportions, i.e., 1 in 5, they give rise to symptoms.

Age Incidence.—Unknown in children. Common in mid-life—30–50. Commonest in old age—but often latent.

Great frequency and latency in old age are probably due to degeneration of muscular tissue of gall-bladder and ducts.

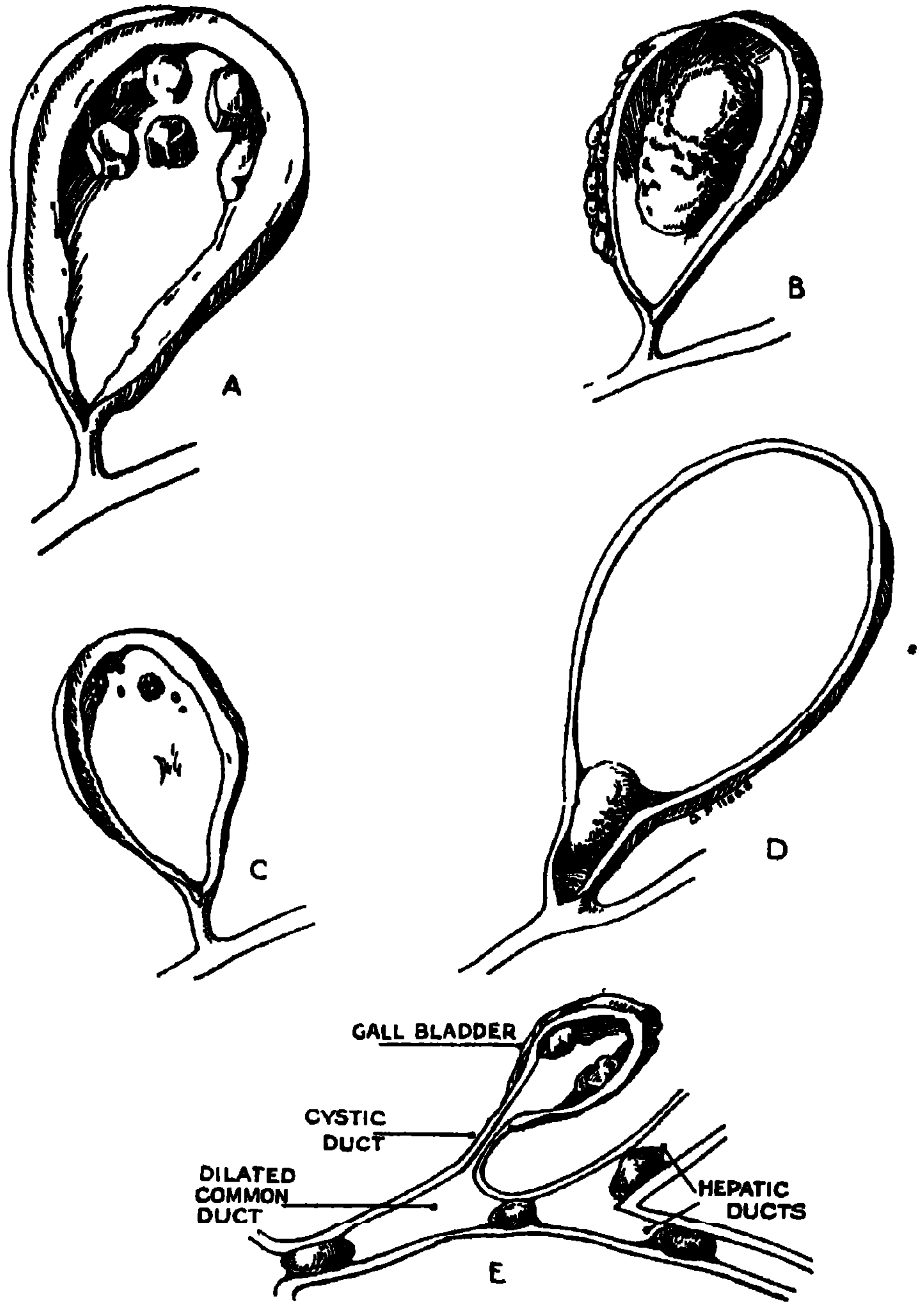


Fig 176—Diagram of various conditions of gall stones and gall bladder **A**, Cholecystitis, with multiple faceted stones and thick gall bladder, **B** Single cholesterin stone, **C**, Gall bladder with small mulberry stones **D**, Single stone impacted in cystic duct, gall bladder dilated, **E**, Stones in ampulla of Vater, common duct hepatic ducts, and gall bladder The shape of the gall bladder is diagrammatically simplified, Hartmann s pouch not being shown

Gall-stones, *continued.*

Symptoms from the presence of gall-stones vary according to their site, the presence of infection, and according to whether they are stationary, or, having moved, become impacted.

GALL-STONE COLIC only occurs with a stone in a duct, and then with the movement or impaction of the stone. The colic consists in a sudden agonizing attack of pain beginning in the hypochondriac or epigastric region and radiating towards the umbilicus and right scapula. It lasts from a few minutes to several hours; it is accompanied by collapse, feeble pulse, clammy skin, and, towards the end of the attack, vomiting. There may be repeated attacks within a few hours.

JAUNDICE occurs by blocking of the hepatic or common ducts. The presence of a stone in these ducts does not necessarily cause obstruction; it is when infection, causing swelling of the mucous membrane, or impaction of the stone, occurs that complete obstruction followed by jaundice results The jaundice occurs 12 to 36 hours after an attack of colic, and usually disappears 2 or 3 days after. The retention of bile pigments is shown first by appearing in the urine, causing it to be of dark brown colour; later the sclerotics of the eyes become yellow, and then the skin. The fæces become clay-coloured. It is said that jaundice occurs in only 15 per cent of cases in which there are stones in the cystic duct, and only in 40 per cent of cases of stones in the common duct.

TEMPERATURE.—Sometimes rigors, with temperature of 103° or more, occur after colic. Probably due to septic infection at seat of ulceration. Fever may be of an intermittent type (Charcot's intermittent fever), associated with catarrh of the bile-ducts and an impacted calculus. May be remittent and lead on to septicæmia or septic endocarditis: associated with suppuration in bile-ducts.

CALCULI IN GALL-BLADDER.—

a. UNCOMPLICATED.—History of many years of gall-stone dyspepsia. Dull aching pain in epigastrium coming on immediately or shortly after food. Sense of fullness and distension, accompanied by flatulence and belching of wind; this is especially brought on by foods such as pastry, cheese, etc. There are often no physical signs. The patient is often stout and past middle age.

b. COMPLICATED BY ACUTE CHOLECYSTITIS.—After many years of above symptoms, attacks of more acute pain occur. These commence in right hypochondrium and epigastrium, and radiate to the back and shoulders Onset frequently with mild shivering attack, often associated with pyrexia. Vomiting is common. The pain after meals is often increased, and may occur at night and be relieved by food, thus simulating duodenal ulcer.

Physical Signs.—There is tenderness over the gall-bladder. This is best elicited by Murphy's sign, in which with the patient in a sitting position the surgeon hooks his fingers upwards under the costal margin on each side. On the patient taking a deep breath the tender gall-bladder causes a sudden reflex inhibition of respiration. Boas' sign is the presence of an area of superficial tenderness from the transverse processes of the vertebræ to the posterior axillary line between the 11th dorsal and 1st lumbar spine levels.

c. COMPLICATED BY SUPPURATIVE OR GANGRENOUS CHOLECYSTITIS.—The symptoms may commence as an acute cholecystitis and instead of clearing up in two or three days may progress, or the onset may be abrupt with severe pain in epigastrium with vomiting, rigors. pyrexia, and marked tenderness of the upper right rectus muscle. The condition resembles an acute appendicitis, only in the upper right quadrant of the abdomen instead of the lower. On examination, if the local tenderness permits, a mass may be felt in the right hypochondrium consisting of a distended gall-bladder wrapped in great omentum. If surgical treatment is not adopted, perforation of the gall-bladder with general peritonitis may ensue.

CALCULUS IN CYSTIC DUCT.—

After an attack of acute biliary colic, the gall-bladder becomes distended with mucus and may grow to a large size with but few symptoms. If infection occurs, the signs will be those of acute cholecystitis with the presence of a distended palpable gall-bladder. This gives rise to empyema of the gall-bladder. Jaundice only occurs from associated catarrh or obstruction of the common or hepatic ducts.

CALCULUS IN COMMON DUCT.—

There is usually a history of previous attacks of biliary colic followed by transient jaundice. When a stone becomes impacted in the common duct, the jaundice rapidly becomes intense, with itching and pale motions, etc. Whilst at first complete, the obstruction later diminishes, so that there is a variation in the degree of jaundice from day to day This variation is of great importance in diagnosis of jaundice from obstruction due to carcinoma. Infection soon ensues with a stone in the common duct, and rigors and intermittent fever from infective cholangitis occur

Physical Signs.—

ENLARGEMENT OF LIVER.—Slight—Tender.

ENLARGEMENT OF GALL-BLADDER at ninth costal cartilage. Smooth and rounded Points to right pubic spine. Moves with respiration. Can be moved laterally. May be due to mass of calculi, mucus, or pus.

This enlargement of the gall-bladder indicates blocking of the cystic duct, and there is in this case only slight jaundice

WHEN THE COMMON DUCT IS BLOCKED, and in most chronic or recurrent cases, the gall-bladder is contracted.

If a stone produces marked jaundice it does not cause enlargement of the gall-bladder, and vice versa.

Courvoisier's Law.—" When the common duct is obstructed by a stone, dilatation is rare because of chronic disease of the gall-bladder; when the duct is obstructed by other causes, dilatation is common." May be of importance in diagnosis. Absence of enlargement with gall-stones is ascribed to previous inflammation causing adhesions and fibrosis.

COROLLARY TO THE LAW.—

In jaundice due to gall-stones the gall-bladder is small; when due to carcinoma it is usually enlarged.

EXCEPTIONS TO THE LAW.—

1. Where there is a stone or stricture of the cystic duct causing hydrops or empyema, together with the acute impaction of a stone in the common duct.
2. Where there is a stone in the cystic duct pressing upon the common duct.

Where there is distension of the gall-bladder by an acute inflammatory process with obstruction of the common duct by a stone.

Gall-stones—Courvoisier's Law, ***continued.***

4. Where there is chronic induration of the head of the pancreas with a stone in the common duct.
5. Where there is malignant disease of the common duct at any part of its course, or cancer of the head of the pancreas and a chronic sclerosing cholecystitis.

Diagnosis.—Usually follows from association of colic with jaundice, and there may be palpable gall-bladder or stone in fæces.

RENAL COLIC.—Pain radiates into pelvis and thigh; vesical irritation, crystals in urine.

LEAD COLIC.—Chronic constipation, blue line, etc.

GASTRIC ULCER.—Relation of pain to meals.

CANCER OF HEAD OF PANCREAS.—Fixed tumour present, loss of flesh, deep jaundice associated with enlargement of gall-bladder.

MEMBRANOUS COLITIS.—Casts and shreds of the colon are passed.

ABSCESS OF LIVER, ASCARIDES, HYDATIDS.

CHOLECYSTOGRAPHY.— The gall-bladder can be demonstrated by X rays after the ingestion of sodium tetra-iodophenolphthalein The dye is given at 5 p.m. after a fat-free evening meal. X-ray films are taken at 9, 11, 12 noon next day, and a further film taken after a fatty meal to see the rate and degree of emptying of the gall-bladder. Diseased gall-bladders fail to cast a shadow or at the most give a very poor one. Distortion of the gall-bladder may occur from the presence of calculi.

Clinical Groups.—(1) Those in which the stone passes and gives rise to colic and transient jaundice; (2) Those in which the stone becomes impacted and colic is not present, but further sequelæ result.

Impaction: Its Complications and Sequelæ.—

1. IMPACTION OF STONE WITHOUT OTHER COMPLICATIONS.—
   In neck of bladder, or cystic duct: Gall-bladder forms large fluid tumour.
   In common bile-duct: Permanent jaundice, unless fistulous communication with gut is formed
2. IMPACTION FOLLOWED BY SEPTIC SEQUELÆ.—Cholangitis and cholecystitis with rigors. Empyema of gall-bladder. Hepatic abscesses. Perforation of bile-ducts and abscesses in neighbourhood. Passage of stone into peritoneal cavity. Septic peritonitis
3. IMPACTION FOLLOWED BY ADHESION TO STOMACH OR GUT, with formation of fistula and passage of stone. Often no symptoms.
4. IMPACTION FOLLOWED BY ULCERATION AND INTESTINAL OBSTRUCTION.
5. OBLITERATION OF GALL-BLADDER.
6. ULCERATION FOLLOWED BY CICATRIZATION AND STENOSIS OF DUCTS.
7. CARCINOMA resulting from irritation of stone.
8. CHRONIC PANCREATITIS from impaction in the ampulla of Vater.

Treatment.—

MEDICINAL AND DIETETIC.—

FOR COLIC.—Morphine, gr. ¼, cum atropine sulph., gr. $\frac{1}{100}$; hot bath; large quantity of water (warm), with sod. bicarb. and sod. salicyl., to promote flow of bile; inhalation of chloroform.

FOR PREVENTION OF STONES.—Exercise. Diet.

SURGICAL TREATMENT is called for in: **Enlarged gall-bladder—Persistent jaundice—Recurrent attacks of colic—Intermittent fever—Signs of local inflammation—Peritonitis.**

Stones in Gall-bladder.—Cholecystectomy, i.e., removal of the gall-bladder, is the operation of choice. It removes the diseased gall-bladder, makes recurrence of the stones less likely, and greatly shortens convalescence. Cholecystotomy, with drainage of the gall-bladder after removal of stones, should be reserved for cases with acute suppuration and dense adhesions, or for patients in bad condition in whom the larger operation would be dangerous.

Stone in Cystic Duct.—Push back and remove through the gall-bladder, or incise wall of duct, remove stone, and sew up.

Stone in Common Bile-duct—Push forward or back, or excise and sew up. If the stone is in the third part of the duct, this must be done by opening the second part of the duodenum and removing the stone through the posterior wall of the gut

It is advisable to drain down to the duct, but seldom necessary to insert a tube into it.

BILIARY FISTULA

External.—

CAUSES —Gall-stones—Penetrating wounds of gall-bladder or ducts—Hepatic abscess (especially of traumatic origin)—Surgical treatment of cysts or abscess of liver—Cholecystotomy—Actinomycosis—Malignant disease—Congenital.

POSITION —Usually at umbilicus or right hypochondrium. May be in hypogastrium or inguinal region.

SYMPTOMS.—Discharge of bile, mucus, blood, or pus Quantity will depend on patency of ducts. If entire bile is discharged externally, patient emaciates and dies

PROGNOSIS.—When due to malignant disease, abscess in depths of liver, or impermeable stricture—bad. Otherwise—good.

TREATMENT.—Keep sinus clean. Drain. Scrape and pack. Remove gall-bladder Remove obstruction from common duct. Cholecystenterostomy.

Internal.—

CAUSED by adhesion to and ulceration into . Stomach—Colon—Duodenum—Pleura—Pelvis of kidney—Portal vein—Encysted peritoneal cavities. Or by malignant infiltration of same organs

SYMPTOMS.—Gall-stones may: (1) Be vomited, (2) Be voided per anum; (3) Cause intestinal obstruction; (4) Cause encysted peritoneal collection of fluid.

ABSCESS OF THE LIVER

Causes.-

1. TRAUMATISM.—Chiefly pyogenic cocci. Blows on hypochondrium. Penetrating wounds: From outside—From stomach (fish-bones, pins).
2. GALL-STONES.—Chiefly *B. coli communis.* Direct ulceration from gall-bladder or ducts Indirect infection, especially when stone ulcerates in the common duct.
3. PARASITES —Ascarides—Creep up duct from duodenum—Especially in children—Die and act as septic foreign body. Hydatid cyst. Distoma (very rare). Coccidia—Small multiple abscesses.

Abscess of the Liver—Causes, ***continued.***

4. DIRECT EXTENSION from neighbouring inflammatory foci. Right empyema, peritonitis, right perinephritis, etc.

5. PYÆMIC CAUSES.—

Systemic, via the Hepatic Artery.—Occurring as part of a general pyæmia. Especially in septic inflammation of bones (cranium commonest), ulcerative endocarditis, septic diseases of lungs.

Portal, via the Portal Vein (pyogenic cocci, *B. coli communis, Amœba coli,* tubercle, actinomycosis).—Ulcers of stomach or duodenum—Appendicitis—Operation for piles—Septic inflammation of umbilical cord, especially in the newborn.

Tuberculous abscess of the liver is a single ragged cavity, and is generally secondary to hypertrophic tuberculous disease of the cæcum.

Actinomycotic abscess is honeycombed.

6. TYPHOID FEVER —Direct extension up bile-ducts By pylephlebitis. Indirectly through a focus of suppuration.

7. TROPICAL ABSCESS.—

Geographical Distribution.—Commonest in tropics Mediterranean littoral. Occasional in England.

Climate and Season —Greatest moisture, and greatest daily variation in temperature.

Race.—Europeans are more subject than natives.

Sex and Age.—Men to women as 30 to 1. Age 20 to 40.

Alcohol is great predisposing cause.

Malaria may act as a predisposing cause, but tropical abscess often occurs where malaria is unknown, e.g., Chile.

Dysentery.—In 75 per cent of cases. *Amœba coli* found in stools and in the abscesses; often not found in pus at first aspiration, but may be found 2 or 3 days later They are invariably found in scrapings from wall of abscess.

Pathology.—

SINGLE.—Tropical, in 75 per cent—Gall-stones—Ascarides—Spreading from neighbouring suppuration.

MULTIPLE in all other cases; typically the pyæmic.

MAY OPEN into. Lungs—Pleura—Pericardium—Stomach—Intestine—Peritoneum—Right kidney pelvis—Vena cava—Externally.

SITUATION.—Commonest in upper part and back of right lobe.

Symptoms.—Often latent until rupture in tropical abscess

Prominence, or bulging, in hypochondrium. Redness and œdema of skin.

Dull aching Pain over liver. Sharp pain indicates perihepatitis.

Enlargement of Liver —Swelling moves with respiration. Fluctuation is rarely felt. Liver is tender.

Muddy Complexion.—Slight jaundice.

Auscultation.—Friction sounds over liver. Signs of pleurisy at base of right lung. Crepitations at base of right lung. Splashing sounds when abscess has burst into the pleura.

Enlargement of liver dullness, generally upwards. Stomach, colon, and right kidney pushed down. Pain in right scapula and shoulder. Cough (from irritation of phrenic or invasion of lung). *Amœba coli* may be present in sputum. Dyspnœa from encroachment on chest.

TEMPERATURE.—Hectic (with rigors) in cases of injury or pyæmia. Often normal in latent tropical abscess.

DIGESTIVE DISTURBANCES.—Vomiting, diarrhœa, constipation, etc.

Complications caused by abscess bursting :—

INTO STOMACH.—Vomiting of pus.

INTO INTESTINE, generally colon.—Pus per anum.

INTO PERITONEUM.—Fatal peritonitis.

INTO PELVIS OF RIGHT KIDNEY.—Very rare.

INTO INFERIOR VENA CAVA.—Fatal.

INTO GALL-BLADDER —Pus by bowel. May recover.

INTO PLEURA.—Rare—Symptoms of rapidly-formed empyema.

INTO LUNG.—Signs of pleurisy—Cough. Expectoration of foul, dark pus, often much blood—*Amœba coli*. Seventy per cent of tropical cases in which this happens end well, but in pyæmic cases it is fatal.

INTO PERICARDIUM.—Rapidly fatal.

EXTERNALLY.—Between or below the costal cartilages.

Nervous System.—Attacks of melancholia or hypochondriasis. Delirium and coma at end.

Diagnosis.—

LATENCY.—Some cases are latent, and bursting of abscess is first sign.

SPECIAL SIGNS —Pain in right hypochondrium—Pain in right shoulder —Muddy complexion—Jaundice (slight)—Enlarged liver—Raised temperature.

PRESENCE OF DYSENTERY, septic wounds of head, or any focus of suppuration, makes above symptoms more significant.

LOCALIZED PERIHEPATITIS may give pain like abscess, but there is no enlargement of liver, and symptoms are not so severe.

OTHER VARIETIES OF SUBPHRENIC ABSCESS (*see* p. 387).

ASPIRATION BY NEEDLE is justifiable only if positive result leads to immediate operation. It may show *Amœba coli*, hydatid hooklets, coccidia, actinomycosis, or thick pus like anchovy sauce.

Prognosis.—In single or non-pyæmic cases, mortality varies from 50 to 80 per cent.

Treatment.—Freely open after locating abscess with syringe.

If parietes are not adherent, pack sponges round, and sew parietes to liver before evacuation.

Knife of thermo-cautery is best to cut liver.

If abscess be in right lobe above margin of ribs, the thoracic wall must be incised.

If abscess communicates with pleural cavity, part of chest wall must be freely excised, and both cavities drained.

If empyema exists, but does not communicate with liver, then two separate openings are required.

If abscess bursts into peritoneum, open at once, and if adhesions localize the mischief, then evacuation may be successful.

TREATMENT OF AMŒBIC ABSCESS.—

Aspirate through large bone needle, and wash out abscess with quinine, leaving in half as much as pus taken out. Only perform transpleural drainage if aspiration fails to clear up the abscess.

Give emetine bismuth iodide in gelatin-coated capsules internally as soon as the condition is suspected.

HYDATID CYSTS

Distribution.—Common in Australia and Iceland. Commoner in London than in Scotland or provinces.

SITUATION IN LIVER.—Any part or surface may be involved. Hydatids of liver form 57 per cent of hydatid disease.

Symptoms.—

HEPATIC TUMOUR.—On the superior surface extending upward, liver pushed down. On the inferior surface extending downward, liver pushed up. Fluctuation, or elasticity, with 'hydatid fremitus'.

PRESSURE SYMPTOMS.—
Upon lung—Dyspnœa.
Upon digestive organs—Pain, vomiting, etc.
Upon blood-vessels—Œdema, ascites, piles.
Upon peritoneum—Localized peritonitis, pain.
Jaundice is rare—From pressure, or bursting into bile-duct.

RUPTURE —
Into alimentary canal—Spontaneous cure
Into peritoneum—Shock, collapse, urticarial rash
Into pleura or lung—Pleurisy or pneumonia, possible expectoration of cysts
Into pericardium—Rapidly fatal.
Into gall-bladder—Colic and jaundice.
Into pelvis of kidney, bladder, vena cava, portal vein.
Externally—Fistula.

SUPPURATION.—May result from injury, from presence of other septic focus, from pyæmia Forms hepatic abscess with usual signs.

SPONTANEOUS CURE —Parasite dies, fluid is absorbed, inert pultaceous material remains encapsuled. Cyst may become calcified. Cyst may be filled with bile, and then cured.

HÆMORRHAGE into the sac may follow rupture, and may prove fatal.

COMPOSITION OF HYDATID FLUID.—Neutral reaction. Sp. gr. 1006. About 2 per cent solids—salts, proteins, and extractives Often contains hooklets No albumin.

Diagnosis.—Slow-growing hepatic swelling, which : (*a*) is fluid or elastic; (*b*) moves with respiration Aspiration shows hooklets. Complement fixation test. Blood invariably shows an eosinophilia

DIFFERENTIAL DIAGNOSIS.—

1. Solid Enlargments of Liver.—In these: Solid feel Signs of syphilis or malignant disease. Aspiration produces no fluid
2. Simple Cysts (very rare).—Fluid has no hooklets, and is rich in albumin
3. Abscess.—Rapid development. Tender to pressure. Throbbing pain. Rigors and temperature.
4. Enlarged Gall-bladder.—Associated with colic and jaundice. Lateral movement possible.
5. Subphrenic Abscess.—Inferior border of liver displaced but unaltered in outline. Signs of inflammatory process. Aspiration produces pus or gas.
6. Cyst or Swelling of Right Kidney.—Moves slightly with respiration. Colon is in front, not below.

7. Pleural Effusion.—Dullness is more diffuse and less localized. Constitutional symptoms are more marked.
8. Aneurysm of Hepatic Artery or Aorta.—Expansile pulsation. Blowing murmur. Jaundice always when hepatic artery is involved. Pain very great.
9. Ovarian Cyst —Pelvic connections Line of separation from liver.
10. Hydatid Cyst of Abdominal Wall.—Liver felt moving independently.
11. Hydatid of Base of Lung.—Cough with blood-stained expectoration.
12. Phantom Tumour
13. Local Abscess in Peritoneum.
14. Ascites.

Prognosis.—Bad when suppuration or rupture occurs—When cyst has to be reached through thorax.

Mortality after incision through abdomen, 10 per cent After incision through thorax, 29 per cent.

Treatment.—Never aspirate. Expose the cyst, inject 2 per cent formalin to render contents sterile, and then endeavour to shell out the cyst. If this fails, sew the sac to the parietes (i.e , marsupialize), and then incise. Never try to remove the fibrous ectocyst, as excessive hæmorrhage will occur from the liver.

Other Cysts of the Liver.—All rare.

SIMPLE SEROUS CYSTS —May be large or small. Usually no symptoms Fluid becomes solid on boiling No hooklets Probably lymphatic or dilated mucous glands

Treat as if hydatid

MULTILOCULAR CYSTIC DISEASE —Often associated with cystic kidneys. Probably mucoid degeneration of bile capillaries Commonest in old people Generally not recognized.

No treatment

CYSTIC ADENOMA.—Localized glandular tumour containing cysts.

DERMOID CYSTS —Very rare.

MOVABLE LIVER

Causes.—

CONGENITAL.—Elongation of coronary and suspensory ligaments. Absence of coronary ligaments

ACQUIRED.—Enlargement or growth of gall-bladder Narrowing of outlet of thorax (tight lacing). Weight or traction of tumours or cysts. Laxity of abdomen, caused by many pregnancies. Fatty degeneration of suspensory ligament following peritonitis

Symptoms.—Often associated with enteroptosis, movable kidney, or spleen. Dragging pains Occasional jaundice Tumour, shape of liver, in abdomen, which can be replaced in right hypochondrium. Resonance in hepatic region. Difficulty in walking. Digestive derangements. Almost always in women.

Treatment.—Belt. Discontinue corsets. Possibly hepatopexy.

SOLID ENLARGEMENTS OF THE LIVER

Malformation.—A down-growing tongue of hepatic substance, usually connected with the anterior margin of the right lobe. Known as Riedel's lobe.

It is of importance only because it may be mistaken for gall-bladder, kidney, or other tumours

Actinomycosis.—Rarely primary. Usually secondary to an intestinal infection, or an extension from the thorax. The liver is enlarged, with bossy outline, and is associated with signs of abdominal or pleuritic chronic inflammation. It softens and breaks down into an abscess, the pus of which may give the clue

Prognosis is bad and treatment of little avail.

Syphilis.—

DIFFUSE HEPATITIS.—Usually seen in congenital cases The liver becomes hard, nodular, much enlarged from an interstitial fibrosis.

GUMMATA —Occur in acquired or congenital forms They cause a bossy enlargement of the surface, with few or no symptoms unless the portal fissure is encroached upon.

Amyloid Disease may result from syphilis or chronic suppuration.

Angioma is the only innocent new growth which is not rare. May be congenital or acquired, and it usually grows just beneath the capsule.

CONGENITAL CASES form large masses reaching to the umbilicus which are not encapsuled.

ACQUIRED CASES occur at about 60 as multiple encapsuled tumours about ½ to 1 in. across.

Carcinoma, as a primary growth, may arise from the hepatic cells, or from those of the bile-ducts The mass is single Secondary growths are common and multiple.

Sarcoma is very rare primarily, but common secondary to bone and melanotic deposits.

SYMPTOMS OF MALIGNANT ENLARGEMENT.—Nodular enlargement which encroaches upon the thorax and abdomen Jaundice, emaciation, cachexia, and ascites are common, if death does not occur from the primary disease

NEOPLASMS OF GALL-BLADDER AND DUCTS

Carcinoma is common. Sarcoma, papilloma, and fibroma are very rare.

Carcinoma of Gall-bladder.—

PATHOLOGY.—Generally scirrhous. Columnar-celled—from epithelial lining. Spheroidal-celled—from mucous glands. 90 per cent associated with gall-stones. Growth begins in mucous membrane, and at first it is a localized growth.

SYMPTOMS.—

IN EARLY STAGE.—Very vague and indefinite.

IN ADVANCED STAGE.—

Jaundice in 70 per cent of cases. From impaction of calculus or growth, from invasion of ducts, from pressure of glands at portal fissure.

Pain.—Sharp and paroxysmal, or constant and gnawing.

Tumour.—Hard, nodular. Sometimes suppurating.

Loss of weight. Spontaneous hæmorrhages. Gastro-intestinal disturbances.

SEX.—Women are attacked three times more often than men.

AGE.—Between 50 and 60.

DIAGNOSIS from carcinoma of liver secondary to other growth; carcinoma of pylorus or pancreas.

PROGNOSIS.—Life lasts few months to two years.

TREATMENT.—If diagnosed before infiltration of liver has begun, remove gall-bladder and resect adjacent part of liver.

Malignant Growths of Bile-ducts.—Columnar-celled carcinoma. Common bile-duct in duodenum is usual seat, but any other part may be affected.

Begins as a papillary or submucous growth. Constricts duct. Invades pancreas, duodenum, etc. Seldom larger than hazel-nut or walnut.

No special relation to gall-stones. Both sexes equally affected.

SYMPTOMS —Indefinite pains in area of eighth dorsal nerve. Gradual onset of jaundice with all its signs. Hæmorrhages—cutaneous and mucous. Skin becomes olive-green Intense itching. Enlargement of liver and gall-bladder. Slow pulse Cholæmia—drowsiness, vomiting, delirium.

DIAGNOSIS.—

From Impacted Gall-stone.—History of colic. Duration not more than few months in cancer.

From Cancer of Head of Pancreas.—Almost impossible

PROGNOSIS —Seldom live more than three months after jaundice has become established.

TREATMENT.—Cholecystenterostomy is the only possible operation in most cases. Results are bad: hæmorrhage is so very liable to occur in jaundiced subjects, the administration of vitamin K prior to any operation should always take place.

Removal of growth is very seldom possible.

CHAPTER XLII

AFFECTIONS OF THE PANCREAS AND SPLEEN

AFFECTIONS OF THE PANCREAS

Diabetes.—Is caused by total removal or destruction of the pancreas by atrophy or new growth.

Often associated with fatty and fibrous degeneration of pancreas.

Interacinar pancreatitis, with destruction of certain special cells called the islands of Langerhans, is the lesion most frequently found in pancreatic diabetes.

Injury.—Is very rare, and then usually fatal.

A blood-cyst may form in the lesser peritoneal cavity, and in this pancreatic ferments are present

Treat by opening and emptying from in front, with ligature of bleeding vessels and drainage from behind

Pancreatic Calculus.—Very rare. Composed chiefly of carbonates. Causes colic, and is associated with diarrhœa, and often diabetes.

PANCREATITIS

Aetiology.—The relation of the pancreatic duct to the common bile-duct, the two having a common opening into the duodenum, makes any obstruction of the ampulla of Vater (the common opening) by a gall-stone or spasm of sphincter of Oddi likely to produce pancreatic obstruction and inflammation

INJURY by an abdominal contusion or operation may lead to subacute or chronic pancreatitis

BACTERIAL INFECTION —Every case shows organisms present generally of the *B. coli* group Protrypsin plus bacteria results in the formation of trypsin, and this produces rapid necrosis of the pancreas The route of infection is via lymphatics from an infected gall-bladder

Pathology.—

ABSENCE OF PANCREATIC DIGESTION, as shown by: (1) The presence of undigested meat fibres in a patient not suffering from diarrhœa; (2) Abundant undigested fat in the motions, causing the pale stools.

FAT NECROSIS, i.e., a splitting up of fat into glycerin and fatty acids. The latter remain as opaque masses in the tissues, united to calcium salts. It is the result of the diffusion of the fat-splitting pancreatic ferment in the blood. It occurs chiefly in the abdominal fat, both visceral and parietal. It is common in acute and subacute pancreatitis, but rare in the chronic.

HÆMORRHAGE.—Is conspicuous and well marked in the majority of the acute cases. It occurs: (1) In the pancreas itself; (2) Into the peritoneal tissues around. It may be due to the action of the glycerin liberated by the fat-splitting process, and is often increased by coincident jaundice.

CRYSTALS IN THE URINE.—In many cases of obstructive and acute pancreatitis the urine yields a crop of fine yellow crystals when boiled with phenyl-hydrazine after oxidization (Cammidge).

Classification.—

1. ACUTE —
   - *a.* Hæmorrhagic: (i) Ultra-acute, in which the hæmorrhage precedes the inflammation, the bleeding being profuse both within and outside the gland. (ii) Acute; inflammation precedes the hæmorrhage, which is less profuse and is distributed in patches throughout the gland.
   - *b.* Gangrenous.
   - *c.* Suppurative (diffuse).
2. SUBACUTE.—Abscess of the pancreas.
3. CHRONIC —
   - *a.* Interstitial: (i) Interlobular. (ii) Interacinar.
   - *b.* Cirrhosis of the pancreas.

Acute Pancreatitis.—

PATHOLOGY.—Gangrene or extensive hæmorrhage into the pancreas, with marked fat necrosis.

ONSET is sudden, but may be preceded by a blow.

SEVERE PAIN, TENDERNESS, AND SWELLING in the epigastrium.

SHOCK profound, pulse small and quick, temperature subnormal.

SEVERE VOMITING AND MARKED CONSTIPATION —The latter may yield to enemata and give place to diarrhœa.

Case presents signs and course of Acute Peritonitis of an upper abdominal type, and ends fatally in about three days.

CYANOSIS —Bluish colour of whole body.

DIAGNOSIS is seldom possible from ruptured viscus or acute high obstruction.

TREATMENT.—Median incision establishes the diagnosis by finding: (1) The swelling and hæmorrhage in the pancreatic region; (2) The fat necrosis The organ should then be explored and drained by going through the gastrocolic omentum and opening the posterior wall of the lesser sac. If the condition of the patient justifies further interference the gall-bladder should be drained.

Delayed treatment when diagnosis is certain has been tried in some cases Operation is only undertaken if an abscess forms.

Post-operative Treatment.—Administration of saline and protection of skin from autodigestion by a 0·2 per cent hydrochloric acid ointment.

Subacute Pancreatitis.—

LESS SUDDEN IN ITS ONSET, and marked with much less collapse than the acute variety.

VOMITING is not so severe.

Subacute Pancreatitis, *continued.*

DIARRHŒA becomes prominent, blood and pus passed in stools.

A VERY IRREGULAR TEMPERATURE, rising to 103–105° F. in the evening.

LOSS OF FLESH very rapid.

MARKED TENDERNESS.

DEFINITE SWELLING OR ABSCESS may form over pancreas.

THE ABSCESS may track: (1) Up as a subphrenic; (2) Out as a perirenal; (3) Down as an iliac or psoas; or (4) Forwards as a peritoneal abscess.

COURSE.—Case lasts for weeks or months, and may recover.

TREATMENT.—Calomel and salol by mouth. Gastric lavage, and enemata.

OPERATION FOR ABSCESS.—Exploratory from in front. Draining behind or in front, after having packed off peritoneal cavity.

Chronic Pancreatitis.—

CAUSES.—(1) Biliary catarrh or calculi in the termination of the common bile-duct, which also obstruct the duct of the pancreas; (2) Catarrh, ulcer, or sepsis in the duodenum; (3) A pre-existing acute or subacute pancreatitis.

ANATOMY.—Suppurative catarrh of the pancreatic ducts Interstitial inflammation of the gland, with swelling at first and contraction later. Adhesions of duodenum, pylorus, and gall-bladder, with distension of the latter.

ONSET is usually gradual, but it may be sudden, with pain and jaundice.

PAIN may be both continuous and paroxysmal, and in both cases is in the epigastrium above the umbilicus.

JAUNDICE is well marked, and deepens with each paroxysm of pain.

ASTHENIA, with loss of flesh and digestive disturbance, e g., dyspepsia and vomiting, is well marked.

STOOLS are copious, loose, light-coloured, offensive, and contain free fat.

HECTIC FEVER with rigors is not infrequent.

THE HEAD OF THE PANCREAS can be felt as a hard tumour, and often the gall-bladder is distended.

VARIETIES.—

1. Syphilitic, found in the congenital disease in infants.
2. Interlobular. Affects the tissue between the lobules, and the islands of Langerhans escape. It is the common form arising from duct infection, and produces no glycosuria.
3. Interacinar. The inflammatory fibrous tissue penetrates the lobules and invades the islands of Langerhans. It is associated with diabetes.

DIAGNOSIS.—Is usually confused with chronic gall-stone cases, and recognized only during the operation. The tenderness in the midline, and tumour, may make it clear.

CANCER OF THE HEAD OF THE PANCREAS is often indistinguishable from this disease. The onset is in older people, is more painless, and the course more rapid.

COURSE.—May last for years unless glycosuria supervenes.

TREATMENT.—Any calculi in the biliary passages should be removed, and the gall-bladder anastomosed to the stomach or duodenum. This will relieve the jaundice, which is caused by pressure of the head of the pancreas on the common bile-duct.

PANCREATIC CYSTS

CLASSIFICATION.—(1) Retention; (2) Proliferation; (3) Hydatid; (4) Hæmorrhagic; (5) Congenital; (6) Pseudo-cysts.

1. Retention Cysts.—Caused by a calculus, interstitial fibrosis, pressure of a tumour of the duodenum, or kinking. Form the majority of simple cysts.
2. Proliferation Cysts.—Either adenomata, like ovarian tumours, or carcinomata.
3. Hydatid Cysts.—Very rare.
4. Hæmorrhagic Cysts.—May be left after the absorption of blood from a cavity in or around the pancreas.
5. Congenital Cystic disease, similar to that of the kidneys and liver.
6. Pseudo-cysts —Lying in front of the pancreas, often following trauma and inflammatory conditions. Probably are simply formed by the filling of the lesser peritoneal sac with fluid exuded by injury of the pancreas.

ANATOMY.—Usually in the body of the gland and often of large size, holding fifteen to twenty litres. The inner wall is smooth, but in proliferation cysts there are sometimes papillomata Dense adhesions are the rule.

Fluid is dark, viscid, and mucoid, and may contain proteolytic, emulsifying, and amylolytic ferments.

SYMPTOMS —Epigastric pain. Dyspepsia, vomiting, and rapid loss of flesh. Alterations in the fæces or urine (fat or sugar) are exceptional. Absence of pancreatic secretion in the intestine is shown by Sahli's sign, viz , the fact that salol is not decomposed into salicylic and carbolic acids, whose presence in normal cases can be detected in the urine.

SIGNS.—An epigastric elastic swelling rather to the left of the midline. It is usually behind the stomach and transverse colon. The swelling is fixed, and has a transmitted pulsation. It may take four different courses in its growth:—

1. Between the stomach above and the colon below, both lying in front of it. This is much the commonest.
2. Above the stomach, behind the gastrohepatic omentum.
3. Into the layers of the transverse mesocolon.
4. Below the transverse colon.

When it becomes large it fills the whole abdomen. It is usually fixed, and moves very little with respiration.

DIAGNOSIS.—From cysts of the left kidney, liver, spleen, mesentery, omentum, and ovary, and from abdominal aneurysm (palpate in knee-elbow position).

TREATMENT.—

Extirpation, if possible.

Evacuation and Drainage.—Marsupialize first, as fluid has marked digestive properties and must not be allowed to escape into general peritoneal cavity. Protect skin with mineral fat. If cyst cannot be marsupialized, aspirate, pack off, and drain.

NEW GROWTHS OF THE PANCREAS

Carcinoma.—May be scirrhous, encephaloid, columnar, or colloid. Usually in the head of the gland. It spreads thence into the bile-duct, duodenum, colon, ureter, portal vein.

SYMPTOMS.—Pain is gradual in onset, but usually severe. Jaundice, with enlargement of the gall-bladder, is chronic, very deep, and often accompanied by enlargements of the liver. Tumour is felt only in about a quarter of the cases. Rapid wasting, vomiting, copious foul stools containing undigested fat and muscle fibre.

COMPLICATIONS.—In addition to the usual biliary obstruction (1), there may be: (2) Pyloric obstruction; or (3)· Ascites from pressure on the vena cava and portal vein.

DIAGNOSIS.—FROM GALL-STONES, by the painless onset of jaundice, the enlargement of the gall-bladder, and the rapid emaciation.

TREATMENT.—Anastomosing the gall-bladder to the duodenum or stomach will relieve the jaundice. Resection of the head of the pancreas has been successfully performed.

AFFECTIONS OF THE SPLEEN

Congenital Abnormalities.—

ABSENCE very rare

PRESENCE OF ACCESSORY SPLEENS or splenculi in the gastro-splenic or great omentum.

Effects of Removal.—

BLOOD CHANGES —Increase of leucocytes, decrease of red cells, diminution of hæmoglobin. These changes are most marked two or three months after removal.

LYMPHATICS.—The lymph-glands often become enlarged, and lymph-nodes appear in the mesentery and omentum where they did not exist before.

BONE-MARROW.—The activity of the cellular changes in the bone-marrow is greatly increased.

GENERAL —Slight pyrexia, thirst, polyuria, and abdominal tenderness. The effects are less marked in young patients than in old.

Movable Spleen.—

CAUSES.—(1) Elongation or rupture of supporting peritoneal folds; (2) Increased size of the spleen; (3) Glénard's disease; (4) Tight lacing.

RESULTS.—

DISPLACEMENT.—The movable spleen may lie in any position, and may even occupy the sac of an inguinal hernia. Left iliac fossa and floor of pelvis behind uterus are perhaps the positions which most often cause errors in diagnosis.

ROTATION.—Axial rotation often accompanies extreme displacement. It is usually a half turn or one whole turn (180° to 360°). Symptoms are produced similar to those of a twisted ovarian cyst. The splenic vessels become thrombosed, and adhesions usually fix the spleen in the position where rotation has occurred.

ENGORGEMENT AND HÆMORRHAGE.—These may occur in any displaced spleen, but are specially likely in cases where the pedicle is twisted. Bleeding into the splenic substance may produce cysts.

Changes in Other Organs.—The stomach and pancreas are dragged down, and the tail of the latter may form a part of the pedicle. Adhesions may form with any other viscera, especially with uterus and bladder in pelvic displacements.

TREATMENT.—

Splenectomy in the majority of cases, i.e., in all when torsion, hæmorrhage, and adhesions have occurred.

Splenopexy.—The spleen is sewn into a peritoneal pocket made over the inner surfaces of the 9th and 10th ribs.

Injuries of the Spleen.—

PUNCTURED WOUNDS, either by stabs or bullets, are very fatal from hæmorrhage.

Treatment.—Immediate laparotomy. Suture of the spleen in all cases except those of extensive laceration, in which the organ should be removed

SUBCUTANEOUS RUPTURE.—

Causes are similar to those of ruptured liver, but in many cases there is a pre existing condition of malarial or other splenic enlargement.

Anatomy.—The spleen may be lacerated, torn in two, separated from its pedicle, or reduced to a pulp in its capsule The left kidney and left lobe of the liver are often torn at the same time.

Symptoms.—These are exactly similar to those of a ruptured liver, except that both cause and results are more marked on the left side than the right. There is often a well-marked latent period of hours or days between the initial pain and the subsequent signs of internal hæmorrhage. This is caused by clotting of the blood in the splenic vessels. Shifting dullness occurs in both flanks, but whilst the right flank can be made completely resonant, the left does not become so because it is occupied by clotted blood which does not move. If untreated, half the cases die within one hour of injury—the majority die within 24 hours.

Treatment —Rapid exposure and clamping of the pedicle, followed by removal of the spleen, or by gauze packing and suturing in cases of partial rupture.

Delayed Rupture of the Spleen may occur up to 15 days after the initial injury. In this case there has been a partial rupture of the spleen and a subcapsular hæmatoma has formed to close the rupture. This hæmatoma then becomes digested by the pancreatic ferments escaping from the associated injured tail of the pancreas.

Diseases of the Spleen of Surgical Importance.—

ABSCESS.—Is usually metastatic, arising from a septic embolus in the course of an acute fever, e.g., enteric, malaria, endocarditis, or portal pyæmia.

Symptoms.—Rapid enlargement, with abdominal tenderness and rigidity in the left hypochondrium.

Treatment.—Drainage of the abscess if adherent to the parietes. Removal of the spleen if non-adherent.

TUBERCLE.—Very rarely tuberculosis affects the spleen alone and causes enlargement of the organ. In such cases splenectomy has been followed by good results.

CYSTS.—These may be (in the order of frequency) hydatid, hæmorrhagic, serous, or lymph. The signs are those of a cystic tumour in the left hypochondrium, and the treatment is splenectomy.

Affections of the Spleen, *continued.*

Tumours and Enlargements of the Spleen.—

GENERAL CHARACTERS.—A mass in the left hypochondrium appearing at tip of 9th costal cartilage. A sharp notched edge can often be felt. Moves with respiration. Dullness to percussion in front, but not behind. The upper margin disappears under the left costal margin, and is continuous with an area of thoracic dullness underlying the 9th, 10th, and 11th ribs behind.

NEW GROWTHS.—Primary new growths are generally rapidly growing lymphosarcomata. Form nodular tumours which may be painful. Treatment by removal.

PERNICIOUS ANÆMIA.—In this disease removal of the spleen tends to check excessive hæmolysis. Splenectomy should be preceded by careful 'step-ladder' transfusions of whole blood and removal of any septic foci in teeth, gall-bladder, or appendix. It will not cure the disease, but it prolongs life.

LEUKÆMIA.—Removal of the large spleen is usually fatal. But if the size of the spleen is first reduced by repeated applications of radium, then splenectomy has only a mortality of about 5 per cent. It is not clear, however, that life is definitely prolonged.

SPLENIC ANÆMIA OR BANTI'S DISEASE.—A chronic disease, with anæmia and leucopenia, associated with great enlargement of the spleen and a great tendency to hæmorrhage, especially from the stomach. In late stages, cirrhosis of the liver, jaundice, and ascites. The large spleen is often densely adherent to its surroundings. Splenectomy should be done as early as possible after preliminary radium treatment.

HÆMOLYTIC JAUNDICE.—A condition of jaundice without bile in the urine or clay-coloured fæces. May be congenital or acquired. Caused by excessive hæmolysis in the spleen. Blood-cells show excessive fragility in their reaction to salt solution. Splenectomy produces a permanent cure.

CHAPTER XLIII

AFFECTIONS OF THE KIDNEYS AND URETERS

CONGENITAL ABNORMALITIES

Renal Ectopia.—The kidney may be arrested at any stage of its ascent from the pelvis to the lumbar region. Such kidneys are often poorly developed and are very prone to disease. They are distinguished from ptosed kidneys by short ureter and frequently abnormal blood-supply. The kidney may be felt per rectum when pelvic, or if higher up as an obscure abdominal tumour.

Malformation.—Division into two or many lobes, such as is normal in many animals, is comparatively common, and such kidneys are more liable to calculus formation.

DOUBLE URETER, which may affect only the upper part or the whole length to the bladder, is fairly common.

SUPERNUMERARY VESSELS are common. The accessory renal artery usually comes off separately from the aorta below the main renal artery, and it may form a band over which the ureter becomes kinked, thus bringing about hydronephrosis.

Abnormalities in Number.—

SOLITARY KIDNEY —Occurrence: Approximately 1 in 2000 cases. The kidney may be absent or developmentally insignificant, being replaced by a fibrous mass and the ureter by a fibrous cord. There is a compensatory hypertrophy of the opposite kidney.

FUSION OF THE TWO KIDNEYS INTO A SINGLE MASS.—This is the most important of all the serious forms of congenital abnormality. It usually forms a horse-shoe viscus, the two kidneys being united at their lower pole, and generally being lower than normal. They may form a disc or S-shaped organ in one lumbar region or in the pelvis; this is known as crossed renal ectopia. Unlike the conditions when one kidney is atrophied or absent, it gives no evidence by cystoscopic examination, as the ureteric orifices are normal.

Cystic Disease, Sarcoma, and Hydronephrosis.—These conditions are occasionally congenital.

MOVABLE KIDNEY

Degrees of Mobility.—Vary considerably, the kidney usually moving up and down, occasionally forwards as well, pushing the peritoneum in front of it. Ureter is of usual length and there is a kinking of its upper third as the kidney drops.

Movable Kidney, ***continued.***

Aetiology.—Women form 80 per cent of cases. Right kidney affected twelve times as often as left. Age at which symptoms are most marked, thirty to forty-five.

OFTEN FOLLOWS: Repeated pregnancy—Rapid emaciation which deprives the kidney of some of its fat capsule—Tight lacing, pushing down the liver and kidney—Fluxional hyperæmia associated with menstruation—Blow on the loin, but relationship to trauma is doubtful—Any enlargement of the kidney.

OFTEN ASSOCIATED WITH: Dilatation of the stomach—Enteroptosis—Melancholia.

Anatomy.—Scarcity or absence of perinephric fat. Laxity of peritoneum. Kidney is large, soft, and flabby. Renal vessels elongated.

Complications.—Dilated pelvis and hydronephorosis. Frequent kinking of the ureter. Pyelitis. Calculus, tumour, or other disease. Lower pole is tilted forward and inward.

Physical Signs.—Bimanual palpation can grasp the lower pole of the kidney. Abdominal tumour is formed when the kidney 'floats'. It is generally just at the side of the umbilicus. Abnormal resonance in the loin.

Symptoms.—

ASYMPTOMATIC.—Discovered accidentally during a routine examination.

SYMPTOMATIC CASES.—

1. Pain of dragging nature in loin, worse on standing and during menstruation. Pain relieved by recumbency.
2. Attacks of renal colic due to kinking of the renal pelvis, or rotation of the renal pedicle, causing a temporary hydronephrosis.
3. Attacks of pseudo-biliary colic, when the right kidney is affected Pressure and dragging on the duodenum causes temporary kinking of the bile-duct with associated transient jaundice.

NEURASTHENIC TYPE —Marked hypochondriacal symptoms present. On no account should operation be performed in this type of case (unless progressive dilatation of pelvis is occurring).

Diagnosis.—

FROM OTHER KIDNEY DISEASES, e.g., calculus, tubercle, or new growth. Here some distinct feature, e g., crystals, bacilli, or hæmaturia, is present, and the course is more definite and rapid.

FROM OTHER FORMS OF CHRONIC ABDOMINAL PAIN.—Ovaritis or prolapsed ovaries. Uterine displacements. Recurrent appendicitis. Colic and constipation.

FROM OTHER ABDOMINAL SWELLINGS.—Abdominal right lobe of the liver. Gall-bladder distension or growth. Abnormal spleen. Growths of the cæcum. Ovarian tumour.

The fact that the kidney can be replaced in the loin, and the sickening pain felt on pressure on the kidney, are the main facts which prove a swelling to be displaced kidney

Prognosis.—

PROSPECT OF CURE.—After inflammatory attacks. If the patient becomes fat. Sometimes after pregnancy.

PROSPECT OF BECOMING WORSE.—When associated with enteroptosis or hydronephrosis.

PROSPECT OF DANGER.—When associated with occurrence of renal crises; hydronephrosis or pyonephrosis.

Treatment.—

PALLIATIVE.—Indicated in: (1) Mild cases; (2) Cases associated with hysteria or hypochondriasis, (3) General enteroptosis.

CONSISTS OF: Rest; fattening diet. Belt with pad over kidney region; must be applied when the kidney is replaced.

RADICAL.—

1. NEPHROPEXY.—When symptoms are severe and not relieved by rest and belts. When hydronephrosis is present. When renal crises occur.

   Kidney is slung to the lower intercostal spaces by the partly detached capsule or a strip of fascia lata or ribbon catgut. Always fix to the diaphragm, otherwise movements of latter will pull it down again.

   Results: 1–2 per cent die; 90 per cent are cured of the pain; 50 per cent are cured of gastro-intestinal symptoms.

2. NEPHRECTOMY.—Only when the kidney is disorganized by hydronephrosis or other disease.

INJURIES OF THE KIDNEY

SUBPARIETAL OR NON-PENETRATING INJURIES

Causes.—Direct violence; crushes. Indirect violence; impact of ribs; muscular violence can rupture abnormal kidney (e.g., hydronephrosis).

Occurrence.—Most commonly in men, and on the right side.

Anatomy.—Any of the following lesions may occur: Fatty capsule torn —perinephric hæmatoma Peritoneum torn—especially in children. Subcapsular hæmorrhage Rupture of the parenchyma, kidney usually splitting along the line of the uriniferous tubules—hæmatoma. Rupture of pelvis—bleeding and extravasation. Rupture of vessels. Thrombosis of vessels. Total pulping of kidney. Tearing of the kidney from its hilum.

Symptoms.—

COLLAPSE —Long and profound. Often delayed when it is due to hæmorrhage. Vomiting

ECCHYMOSIS.—In kidney region. This is a late sign and is rarely seen.

PAIN —In loin, radiating to testis and thigh. Pain and retraction of testis when clot is in the ureter.

TUMOUR.—In kidney region. Painful and ill-defined. Peri-renal hæmatoma.

HÆMATURIA.—Commonest and most characteristic sign.

IT RESULTS FROM: (1) Rupture of the kidney; (2) Simple contusion or congestion; (3) Embolus or thrombosis; (4) Late inflammation; (5) Pre-existing cause, e.g , stone.

IT MAY BE ABSENT in ruptured kidney: (1) When rupture is superficial; (2) Vessels thrombosed; (3) Ureter plugged; (4) Kidney torn from the ureter.

Subparietal or Non-penetrating Injuries—Symptoms, ***continued.***

CHARACTERS.—Profuse. Lasts many days, but may be intermittent. May have clot casts of the ureter.

MICTURITION.—Urgent desire for micturition. Pain and difficulty in the act.

OLIGURIA OR ANURIA.—From shock. Injury of solitary kidney. Injury of both kidneys.

RETENTION OF URINE (rare).—Blood-clot in the bladder. Paralysis of abdominal muscles.

PERITONITIS.—When the peritoneum has been torn.

Results of severe injuries.—(1) DEATH from shock or hæmorrhage; (2) PERITONITIS; (3) INFLAMMATION and suppuration; (4) OCCLUSION OF URETERS, with hydronephrosis, pyonephrosis, or atrophy of the kidney; (5) EXTRAVASATION OF URINE; (6) TRAUMATIC NEPHRITIS; (7) PERINEPHRIC ABSCESS, due to bruising of colon and consequent *B. coli* infection; (8) Traumatic pseudo-hydronephrosis.

Treatment.—

SLIGHT CASES.—Rest in bed, morphine, careful collection of urine, noting carefully if bleeding is becoming more severe. Patient kept in bed until all bleeding has stopped. Intravenous pyelogram should be done before discharging patient.

SEVERE CASES.—(1) Bed, treat for shock, morphine, etc. (2) Half-hourly pulse. (3) Save all specimens of urine. (4) Ice-bag to loin. (5) Operation performed if bleeding continues and becomes severe and threatens life; and if kidney irreparably damaged, nephrectomy must be performed. If not, partial nephrectomy, suturing kidney with ribbon catgut over muscle-grafts (6) For a large perirenal hæmatoma, open and drain.

INCISED AND PENETRATING WOUNDS.

Anatomy and Symptoms the same as above, with addition of: Wound—External hæmorrhage—Escape of urine.

Special Dangers.—Involvement of vessels or hilum. Wound of the peritoneum. Septic processes.

Treatment.—

WOUND.—Enlarge a small wound. Pack and drain a large one. Locate and remove any foreign body.

HÆMORRHAGE.—Expose the kidney. Sew, pack, or remove according to the degree of gravity.

PERINEPHRITIS. PERINEPHRIC ABSCESS

Perinephritis does not occur before puberty because there is no perinephric fat until 12 years of age.

Varieties.—Fibrosclerotic—Fibrolipomatous—Purulent.

Aetiology.—

PRIMARY.—Rare, of little importance. Metastatic infection doubtful.

SECONDARY.—Common. Perinephritis invariably accompanies any severe renal infection.

Acute.—In association with severe renal inflammation such as pyonephrosis or local suppurative nephritis. May resolve or become chronic. Suppuration common (perinephric abscess).

Chronic.—Fibrosclerotic and fibrolipomatous types. Tend to obliterate extrarenal pelvis and invade hilum of kidney (nephrosclerosis). May accompany any chronic renal infection, including tuberculous disease, but stone is the commonest predisposing cause.

Pathology.—

The Abscess varies from small to very large. Often gangrenous walls.

Pus is Fæcal, or with fæcal odour. Softening and inflammation of kidney. Pus is usually behind kidney. May bulge peritoneum forward. Often dense adhesion between capsule and adipose tissue.

Pus may Burst into: Colon, small intestine, or stomach, pleural sac or lung (commonest); ureter, peritoneum, psoas, and thence to groin or buttock, loin.

The Non-suppurative Varieties result in dense fibrous adhesions or a fibro-fatty mass round the kidney, which is compressed and itself undergoes fatty degeneration.

Symptoms.—

1. WITHOUT SUPPURATION.—Symptomless.
2. WITH SUPPURATION.—

Obstinate Constipation —

Constitutional Symptoms.—Temperature, 103°–104° F.

Pain deep-seated. throbbing. radiating. and variable Increased by pressure.

Increased Resistance and weight in loin. Dullness to percussion, fluctuation. Redness and œdema of flank.

Retraction of Testis on affected side.

Œdema of Foot and Leg on affected side.

Urine —Exceptionally contains blood or pus.

Diagnosis.—Marked leucocytosis. Severe toxæmia. Flexion of hip due to spasm of psoas muscle. X rays may reveal calculi. Absence of psoas shadow on the affected side. Diaphragm may be elevated and fixed. Excretory or retrograde urography: filling defects and deformities of calices due to pressure of abscess

DIFFERENTIAL DIAGNOSIS —

Lumbago.—No fever or swelling; bilateral.
Nephralgia.—Periodic. Local swelling absent Hysteria.
Organic Disease.—Better outline. No local heat or œdema.
Spinal Disease.—No inclination to one side. Angular curvature.
Morbus Coxæ.—Local signs of hip-joint disease
Sacro-iliac Disease.—Local signs of sacro-iliac joint disease.
Psoas Abscess.—Nearer midline. Spinal symptoms.
Blood Extravasation.—Relation to injury. Absence of fever.
Appendicitis.—Symptoms begin in iliac fossa.
Fæcal Accumulation.
Splenic or hepatic tumour, typhoid fever, empyema, pneumonia.

Prognosis.—Generally grave—30 per cent die. If opened early, good. If abscess bursts into lung, colon, or ureter, there is still a chance. If abscess bursts into pleura or peritoneum, very bad.

Perinephritis, *continued.*

Treatment.—Hot fomentations. Ointment of iodide of lead, etc. Morphine. Aperients, etc.

EARLY AND FREE INCISION, with drainage. When kidney is disorganized nephrectomy should be performed.

INFECTIONS OF THE KIDNEY

Varieties.—(1) Pyelonephritis ; (2) Pyonephrosis ; (3) Suppurative nephritis.

Bacteria.—Chiefly *B. coli communis.*

Predisposing Causes.—

OBSTRUCTIONS in urinary tract—e.g , phimosis, stricture, stone, enlarged prostate, movable kidney.

Direct Causes.—

1. EXTENSION FROM INFECTION ELSEWHERE IN URINARY TRACT.—Cystitis, urethritis.
2. GENERAL CAUSES —Blood infections; pyæmic embolus; irritating drugs; parasites (*Bilharzia, Strongylus gigas*); pelvic inflammations; pregnancy Exanthemata, diphtheria, typhoid.

1. PYELONEPHRITIS

Definition.—Inflammation and suppuration of the kidney substance.

Types.—

A. PARENCHYMATOUS.—

1. ACUTE.—Diffuse blood-borne infection due to the colon group of bacteria. So-called 'Acute Pyelitis.'

   Symptoms and Signs.—High temperature, malaise. Frequency of micturition. Pain and tenderness in loin Often starts with a rigor. Pus in urine.

   Treatment —Responds well to sulphonamides

2. CHRONIC —High temperature. Kidney enlarged and tender. Urine contains epithelial cells, casts, and pus. Condition clears up without suppuration, but bladder and genital tract may be involved

   Treatment.—Chemotherapy with sulphonamides.

B. SUPPURATIVE.—

1. ACUTE.—So-called 'Surgical Kidney'. Infection with pyogenic bacteria, usually preceded by renal disease (calculus). May occur after instrumentation, but is more commonly blood-borne.

   Treatment.—Chemotherapy. Give bland fluids Hot packs to loin.

2. CHRONIC.—May follow acute condition. and its aetiology is similar.

   Treatment.—Chemotherapy. Plenty of bland fluids

2. PYONEPHROSIS

Definition.—Distension of pelvis and calices with pus. Due to retention and infection.

Aetiology.—All causes of hydronephrosis (q.v.), with sepsis added. Especially calculous hydronephrosis. Most common on right side.

Pathology.—Kidney converted into a many-chambered pus sac. Septa between loculi consist of sclerosed parenchyma. Loculi may be separate or communicate with a central cavity. Cavity may be shut off from ureter. Contains pus, urine, and cheesy material. Calculi, either primary or secondary.

ADHESIONS form, and FISTULÆ may follow, into the loin, colon, stomach, or peritoneum.

Symptoms and Signs.—Generally preceded by those of cystitis, pyelitis, or pyelonephritis. Rigors, diarrhœa, and sickness. Hectic temperature. Emaciation and prostration.

TUMOUR.—Enlarged tender swelling felt in loin. May be lobulated and fluctuating, and may vary in size.

PAIN.—Dull ache with acute exacerbations. Tenderness most marked in front.

URINE.—Scanty pyuria with a little blood. Often masked by cystitis.

Diagnosis.—Especially from hydronephrosis, perinephric abscess, pyelonephritis, or tubercle of kidney.

Prognosis.—Depends on condition of other kidney.

Treatment.—

1. REMOVE THE CAUSE, e g., stricture, enlarged prostate, stone in bladder, etc.
2. TREAT CYSTITIS.
3. OPERATION when diagnosis is plain.

OPERATIVE TREATMENT —

1. NEPHROTOMY, with drainage Remove calculus and break down septa
2. NEPHRECTOMY in cases where nephrotomy has failed or when kidney is hopelessly disorganized.

3. SUPPURATIVE NEPHRITIS

Aetiology.—Abscesses of the kidney.

A SEPTICÆMIC KIDNEY.—'Flea-bitten Kidney.' Multiple small infected emboli forming small abscesses Of no surgical importance.

B. SUPPURATIVE FOCAL NEPHRITIS.—'Carbuncle of Kidney.' Aetiology: Solitary bacterial embolus usually from a skin lesion (boil).

Symptoms and Signs.—Dull, aching pain in loin. Local tenderness. Gradual onset of malaise, followed by rigors. Some leucocytosis.

Pathology.—One or more abscesses occupy the kidney substance, resembling the central slough of a carbuncle There may be a perinephric abscess.

Treatment.—Nephrectomy, if not cured with penicillin.

HYDRONEPHROSIS

Causes.—Some form of gradual or intermittent obstruction.

CONGENITAL.—Phimosis Abnormalities of ureter. Abnormalities of the renal vessels.

ACQUIRED —Urethral stricture. Prostatic enlargement. Vesical stone or new growths. Vesical systole (sustained). Pelvic tumours or displacements. Bands or adhesions of peritonitis. Ureteral stenosis from ureteritis. Ureteral calculus, valve, or pocket. Kinking of ureter from mobility. Traumatic obstruction.

Renal Tuberculosis, *continued.*

Aetiology.—

PREDISPOSING CAUSES.—General debility—Local debility.

AGE.—Miliary in young children: rare Caseous in young adults. Rare in aged.

BOTH KIDNEYS generally affected in miliary tubercle. In caseous tubercle: in early stages 85 per cent unilateral, but in late stages 53 per cent bilateral.

CHANNELS OF INFECTION.—

1. ASCENDING (secondary).—Rare.
2. HÆMATOGENOUS (primary).—Commonest.
3. LYMPHATIC.—Rare, from mediastinal lymph-glands.

Pathology and Morbid Anatomy.—

1. ACUTE MILIARY TYPE.—Kidneys studded with miliary tubercles.
2. ULCERO-CAVERNOUS TYPE.—Starts at apex of a pyramid, and hollows out the pyramids from the pelvis outwards.
3. MASSIVE CASEOUS TYPE.—Whole kidney may be converted into a tuberculous pyonephrosis. This is an advanced stage of the ulcero-cavernous type

URETER often blocked It is usually thickened

Symptoms.—

PAIN.—Slight at first—Severe later—Attacks of colic from blocked ureter—Some tenderness on palpation.

TUMOUR.—Moderate size, often due to perinephritis—Ballottement well marked—May be lobulated.

URINE.—Polyuria—Acid—Pyuria and hæmaturia—Urea and phosphates diminished—Great variations due to blocking of ureter.

PYURIA.—Dirty grey pus in small quantity—Mixed with caseous masses and phosphatic debris.

HÆMATURIA —Sometimes an initial symptom—Slight in amount—Sometimes clots moulded in ureter

BACILLI.—Tubercle bacillus in small numbers and difficult of detection. Septic organisms become abundant late in the disease. Absence of other organisms in acid purulent urine points to tubercle When a direct smear is negative, it is advisable to culture the urine and inoculate a guinea-pig for confirmation of the disease

CYSTOSCOPY.—Ureteral orifice is red, with pouting, swollen lips, or retracted. Methylene blue given as a hypodermic is not excreted, or excreted very slowly, by the diseased kidney.

PER RECTUM OR VAGINAM.—The thickened ureter may sometimes be felt

ALBUMINURIA.—Always present when pelvis is diseased.

POLYURIA.—Very marked, especially at night Is an early symptom.

DYSURIA.—Pain in neck of bladder. The earliest symptom. Sometimes due to phosphatic debris in bladder.

CONSTITUTIONAL.—Evening temperature raised to 100°–101° F.; if secondary sepsis has occurred, to 103°–104° F. Loss of flesh. Anæmia. Rigors and sweats in last stage

THE OTHER KIDNEY may be enlarged and tender from hypertrophy without actually being diseased.

INTRAVENOUS PYELOGRAPHY.—The outline of the pelvis is irregular and has a 'moth-eaten' appearance. *Ascending or instrumental pyelography is only justifiable in doubtful cases.*

Complications.—Tuberculous infection of: (1) Opposite kidney in 25 per of cases; (2) Vesiculæ seminales, vas, epididymis; (3) Bladder and other organs. Varying degrees of perinephritis, with great thickening.

Diagnosis.—

FROM PYELONEPHRITIS.—History of case. Thickening of ureter. Presence of tubercle bacilli.

FROM CALCULUS OF KIDNEY.—Constitutional condition. Vesical irritation. Hæmaturia occurs during rest, and is slight. Tubercle bacilli found on examination, especially by inoculation experiments. Radiograph negative

FROM NEW GROWTHS.—Hæmaturia is much less abundant. Constant pyuria.

Diagnosis as to which Kidney.—Side of tenderness, pain, or tumour. If there is intermittent pyuria it is evidence that one kidney is healthy Indigo-carmine test.

Prognosis.—In unilateral cases, one-third are cured; one-third have persistent manifestations (e g., vesical or genital tuberculosis); one-third die within five years. Occasionally undergoes spontaneous cure. Operative prognosis is about 4 per cent mortality. Ultimate result in successful cases is very good

Treatment.—

GENERAL.—Diet, climate, etc

OPERATIVE TREATMENT.—

1. NEPHRECTOMY.—Where opposite kidney is sound. Operation of emergency for hæmorrhage, colic, pyonephrosis Useless when advanced tubercle exists elsewhere.
2. NEPHRO-URETERECTOMY.—When ureter is diseased
3. PRESACRAL NEURECTOMY in cases of intractable bladder pain.
4. URETERIC TRANSPLANTATION (skin or bowel) is of great value where systolic bladder persists after nephrectomy and progressive hydronephrosis of remaining kidney develops.

RENAL CALCULUS

Structure.—

PRIMARY.—	Uric acid	In acid urine.
	Urates of soda or ammonia	
	Oxalate of lime	
	Phosphate of lime	In alkaline urine.
	Carbonate of lime	
	Cystin	In acid urine, but all rare.
	Xanthin	
	Indigo	

Renal Calculus—Structure, ***continued.***

SECONDARY to urinary infection with urea-splitting bacteria (*Proteus*, staphylococci).—

Mixed phosphates Phosphates of ammonia and magnesia Carbonate of lime	From alkaline urine.

LARGE CALCULI.—Consist of secondary deposits on primary nuclei.

NUCLEUS.—Urate of ammonia when formed in infancy. Uric acid, in adults. Oxalate of lime, after forty. Bacteria—only exceptionally.

NUMBER.—Oxalate generally single. May be up to 200.

PROPORTION OF DIFFERENT VARIETIES.—Uric acid in 75 per cent of all calculi. Oxalate of lime in the majority of those removed, because they cause more suffering.

APPEARANCE AND CONSISTENCE (*Figs.* 179 and 181).—
Uric acid—hard, smooth, fawn-red.
Urates—light yellow, soft, friable.
Oxalates—hard, rough, dark.
Phosphates—chalky, mortary.
Carbonate of lime—round, white, hard
Cystin—soft, crystalline, yellowish green.
Xanthin—smooth, hard, cinnamon-red.

Situation.—Pelvis, calices, ureteral orifice, or parenchyma.

Aetiology.—Unknown. In both kidneys in 25 per cent of all cases.

AGE.—Found at all ages. Clinically very rare below ten years; common between twenty and fifty.

SEX.—Slight preponderance of males.

HEREDITY.—In uric acid stone there is generally a well-marked family history.

SIDE.—Right and left equally affected.

HABITS.—Sedentary life and rich diet.

WATER —Lime in drinking water.

CLIMATE.—More frequent in hot countries, because evaporation and sweating concentrate the urine.

INJURY.—May give rise to calculus round a blood-clot, or first give symptoms by moving a calculus that has been present.

Fig. 179.—Branching renal calculus removed from pelvis of kidney. (× ½.)

Pathogenesis.—

URIC ACID.—Especially before tenth and after fortieth year. Derived from nuclein. Acidity of urine. Rich diet, little exercise. Hepatic congestion. Gout. Pernicious anæmia, leukæmia.

URATES.—Especially in children. Concentrated urine, as in febrile conditions. Dyspepsia.

OXALATE OF LIME.—Commoner in men than women. Nervous, irritable temperament. Studious, sedentary habits. Malaria. Rhubarb, gooseberries, tomatoes, sorrel. Poor, vegetable diet. Excess of acid. Formed often from uric acid. Much lime in water is excreted as oxalate.

PHOSPHATE OF LIME AND PHOSPHATE OF MAGNESIUM.—Due to presence of fixed alkali in urine. Excess of alkaline food. Excess of lime in food. Granular kidney. Phthisis. Dyspepsia.

AMMONIO-MAGNESIC PHOSPHATE —Due to inflammation producing ammoniacal urine.

CARBONATE OF LIME.—Same conditions as phosphates. Especially associated with pus.

CYSTIN.—Contains sulphur. Runs in families.

AGGLUTINATION OF CRYSTALS to form a stone can only take place in presence of a colloid substance, e g , colouring matters of urine, mucus, pus, or albumin.

Pathology of Calculous Kidney.—

ASEPTIC CHANGES.—

NEPHRITIS.—Desquamative, interstitial.

Hypertrophy first. Atrophy later (1) from sclerosis, (2) from pressure of hydronephrosis. Hydronephrosis. Partial hydronephrosis. Increase and induration of fibro-fatty tissue. Kidney converted into lipomatous mass.

SEPTIC CHANGES.—Suppurative pyelonephritis, with secondary calculi. Pyonephrosis. Perinephritis and perinephric abscess External or internal fistula

CHANGES DUE TO PASSAGE DOWN URETER.—Impaction, ulceration, etc.

Symptoms.—

COLIC —

CAUSED BY: Passage of stone to bladder—symptoms will cease unless second stone forms or only a fragment of first is passed. Impaction of stone in pelvis. Passage of blood-clot or mucopus.

PAIN shoots down leg, testicle, groin, or bladder Is paroxysmal.

RIGOR, VOMITING, perspiration, collapse.

URINE is scanty, blood-stained. Frequent micturition, often scalding.

Testicle is retracted and becomes tender.

Attack ends suddenly.

Stone may be passed soon or impacted in urethra

PAIN OF A CONSTANT CHARACTER.—In loin, abdomen, in course of ureter, testicle, thigh, inner side of leg or foot. Dull ache up to lancinating pain. Much worse after movement. Often worse at menstrual periods. Uric acid—least pain. Oxalate—acute pain. Phosphates, great and unremitting pain.

Renal Calculous—Symptoms, ***continued.***

REFLEX PAINS.—In opposite kidney. In bladder—frequent and painful micturition. In uterus and ovary (especially of same side). In testicle. In gastro-intestinal tract—colic, vomiting, and nausea.

HÆMATURIA.—Almost always present (microscopically if not otherwise). Often associated with pain or colic—generally profuse for two or three days after colic ceases. Caused or increased by movement. Abated or stopped by rest.

PYURIA.—Generally only slight or microscopical. Urine is acid, and pus deposits readily

URINE.—Hyaline casts—Slight albumin—Pus, blood, crystals.

PHYSICAL EXAMINATION.—Can be felt in thin subjects very rarely. Tenderness and pricking pain on bimanual examination. Greater resistance of muscles on affected side.

URETERAL CATHETER and CYSTOSCOPE show: Œdema of one ureteral orifice. Difference of urine on two sides.

RADIOGRAPHY.—Oxalates give best shadows. Phosphates give dark shadows. Pure uric acid and urates give faint shadows.

Diagnosis.—

I. FROM OTHER RENAL DISEASES.—

1. RENAL TUBERCULOSIS —In this there are: Pain and hæmaturia, not influenced by rest. Colic occurring only when mucopus is passed. Tubercle bacilli in the urine, or other tuberculous lesions.
2. RENAL TUMOURS —In these: Tumour rapidly grows Hæmaturia is much more copious Pain is more constant and less colicky.
3. MOVABLE KIDNEY —In this: The kidney mobility can be felt. There is absence of crystals in the urine. The two conditions may co-exist.
4. URETERITIS OR KINKING or valve of the ureter.—In these: Intermittent hydronephrosis. Absence of crystals. The two conditions often co-exist.
5. CRYSTALLURIA —In this· Hæmaturia, pyuria, or albuminuria are rare and scanty. The symptoms rapidly disappear under suitable treatment with alkalis, piperazine, etc.
6 NEPHRITIS.—In this: Urine is of low specific gravity. Casts are frequent and constant. Crystals are rare. Pulse of high tension, and heart is hypertrophied.

II. FROM DISEASES OF OTHER ORGANS —

1. LUMBAGO AND INTERCOSTAL NEURALGIA.—In these: The pain is related to muscular effort. Generally bilateral. Muscles are fixed when the pain is present Absence of destructive renal signs.
2. SPINAL CARIES.—In this: Fixity, rigidity, tenderness, and pain of the spine. Urinary symptoms, if present, are associated with paraplegia.
3. BILIARY COLIC.—In this: Relation to jaundice. Signs of tenderness or enlargement of the gall-bladder.
4. APPENDICITIS of recurrent variety.—In this: Tenderness over McBurney's point. Tenderness per rectum. Intestinal symptoms.
5. GASTRIC OR DUODENAL ULCER.—In these: Relation to food. Dyspepsia, vomiting.

In all the above, except sometimes in (2), there is an absence of any urine changes.

6. Disease in the Bladder—growth or stone. In these: Vesical irritation. Pain after micturition. Blood comes at the end of micturition. Cystoscope shows the disease.

Prognosis.—

DANGER FROM: Impaction in ureter. Cicatrization and stenosis of ureter. Atrophy of kidney. Hydronephrosis. Suppurative pyelonephritis. Perinephric inflammation, suppuration. Fistula. Affection of opposite kidney. Anuria.

AFTER OPERATION.—Nephrolithotomy: 2 per cent die. Nephrotomy: 10 per cent. Nephrectomy: 8 per cent.

Treatment.—

PROPHYLACTIC.—Correct obstruction, focal sepsis, or other cause (e.g., hyperparathyroidism and bone disease). Diet is of doubtful value except in cystinuria. Vitamin A of value.

Exercise and Fresh Air.

Avoid malt liquors, port, champagne, vinegar or salads containing it, rich meats, excess of butcher's meat, sweetbreads, liver, kidneys.

Plenty of Diluents: Distilled water, Contrexéville or Vichy waters.

DRUGS.—

Alkalis, bicarbonates, potash, soda.
Citrate, tartrate, acetate of potash
Carbonate and benzoate of lithia.
Piperazine, Sidonal, Hexamine } Diuretics, solvents of uric acid, and antiseptics.

OXALURIA.—As above, but also:—

Avoid hard water, green fruits, asparagus, tomatoes, rhubarb, gooseberries, grapes.

Tonics, e.g., iron, strychnine, and quinine.

PHOSPHATURIA.—Treat neurasthenia and dyspepsia. Acids. Tonics.

SECONDARY CALCULI.—Aseptic precautions Avoid mineral waters. Hexamine, etc

PALLIATIVE TREATMENT —For pain and colic: Morphine. Hot bath. Opium and belladonna fomentations. Alkaline drinks.

RADICAL OR SURGICAL TREATMENT.—

Pyelolithotomy.—Operation of choice. Symptoms which justify it are: Hæmaturia, crystals and pus, constant pain or colic.

Nephrotomy.—For calculous pyonephrosis, and all septic conditions complicating calculi.

Nephrectomy.—If kidney is completely destroyed After nephrotomy has been repeatedly necessary. For intractable fistula following nephrotomy.

Partial Nephrectomy.—In suitable cases.

Lumbar incision when calculus is already in a fistula or perirenal abscess.

CALCULOUS ANURIA

Aetiology.—

CAUSES.—(1) Blocking of ureter by small stone; (2) Destruction of kidney by large stone.

IMPLIES either only one functional kidney, or (rare) reflex suppression in the opposite kidney which has been injured.

Calculous Anuria—Aetiology, ***continued.***

POSITION OF IMPACTION.—Renal pelvis 13 per cent; upper end of ureter 60 per cent; middle of ureter 13 per cent; lower end of ureter 20 per cent; bladder (large calculus) 4 per cent.

AGE AND SEX.—May occur at any age or in either sex. Commonest in males over forty.

HISTORY.—Generally: antecedent history of colic. Sometimes: disease of one kidney (other than calculus). Rarely: simultaneous blocking of both ureters; blocking of ureter of single kidney.

IMMEDIATE CAUSE.—Some jolt, jar, or exercise.

Pathology.—On one side there is absence, atrophy, disorganization, or hydronephrosis of the kidney, or an old impacted calculus.

On other side: Enlarged, congested kidney Rarely hydronephrotic. Impacted calculus in pelvis or ureter.

Symptoms.—

ONSET.—Pain (colic) on side last affected—subsides after one or two days. Or dull aching pain lasting throughout.

Rarely onset is insidious and without marked colic or pain.

Ineffectual dysuria, or polyuria. Anuria may be complete, incomplete, or intermittent.

TOLERANT STAGE.—Complete anuria may last seven or ten days without uræmic symptoms.

If anuria ceases temporarily or permanently, a large quantity of low specific gravity urine is passed at once.

Anuria is rarely complete.

Urine passed in intervals is of low specific gravity, poor in urea, light colour. Rarely with blood, albumin, or casts.

Polyuria may occur and delay fatal result.

There may be constant desire to micturate

Depression, insomnia, and digestive disturbances.

WHEN ASSOCIATED WITH HYDRONEPHROSIS.—Length of disease is much greater—lasts fifteen to twenty-five days. Generally history of repeated attacks of colic and anuria. Tumour, etc., can be felt.

URÆMIC STAGE —Pulse full, slow, irregular. Epistaxis and œdema (due to venous stasis). Temperature low.

Profuse perspiration, pruritus, etc. Profuse salivation and expectoration. Constant and profuse vomiting, with constipation and meteorism.

Contraction of pupils. Muscular tremors, rarely amounting to convulsion. Complete depression of body and mind. Incessant restlessness. Drowsiness or semi-coma. Respiration slow and irregular.

Prognosis.—

In cases left to nature, 80 per cent die; 20 per cent recover by passage of calculus.

In cases operated on, 50 per cent recover.

During tolerant stage, i.e., before contraction of pupils and muscular tremors, there is always a chance of recovery.

Prognosis is worse in relapsing cases.

Diagnosis.—

IN STRAIGHTFORWARD CASES.—Renal colic on one side. History of colic on the opposite. Anuria.

IN OTHER CASES.—**One kidney may have been destroyed without pain. Patients may have forgotten which side the colic was at first. Palpation of ureter may show tenderness. Rectal or vaginal examination may feel the stone. Otherwise open loin on side last affected.**

FROM OTHER CONDITIONS.—

Cancer of Uterus, Cancer of Bladder, and other Pelvic Tumours (frequent causes of anuria).—Diagnosis by pelvic examination.

Traumatic Anuria.—By relation to injury.

Anuria Following Operations on lower urinary tract.—Relation to the operation. Rigors, malaise.

Sulphonamide Anuria.

BRIGHT'S DISEASE.—

Anuria is accompanied by headache, vomiting, coma, and convulsions. What urine there is, is dense, bloody, and solid with albumin. Secretion is resumed gradually.

AMYLOID DEGENERATION.—Long history of polyuria, œdema, etc.

HYSTERIA.—Other symptoms.

DRUGS (cantharides, turpentine, mercury).—History of their inception. Gradual onset. Urine bloody, and like Bright's disease.

RETENTION OF URINE.—Is excluded by passing a catheter.

Treatment.—First attempt to catheterize ureter on side last complained of. Operate as soon as diagnosis is clear. Open kidney last affected—On tender side—On side of greatest abdominal resistance.

NATURE OF OPERATION.—To remove stone, if possible, by nephrolithotomy. To establish lumbar fistula if stone cannot be removed. In cases where stone can be located in lower ureter, then ureterotomy

TUMOURS OF THE KIDNEY

Age.—Two periods of life when they are commonest: (1) Under five years; (2) Forty to fifty years.

Varieties.—Hypernephroma 70 per cent; carcinoma 7 per cent; others 23 per cent.

Distinctive Points of Kidney Swellings.—

1. Colon in front of tumour: either resonant or felt as a roll.
2. No space or resonance between tumour and spine.
3. Do not bulge behind, but only cause a fullness of the loin.
4. Absence of any sharp edges.
5. Slight or no movement with respiration.
6. Generally do not cross midline; do not invade pelvis; have a line of resonance between them and liver; except in extreme cases of hydronephrosis.
7. When they touch the anterior abdominal wall they do so about the level of the umbilicus.
8. Varicocele of recent development is frequent
9. Changes in urine or micturition.

Diagnosis from:—

LIVER TUMOUR OR ENLARGEMENT.—**Moves with respiration. Fingers cannot get between it and ribs. No dullness in lumbar region. No intestine in front**

Tumours of the Kidney—Diagnosis, ***continued.***

SPLEEN.—No bowel in front. Sharp edge—sometimes notched. Resonance between it and the spine. Moves with respiration.

SUPRARENAL.—General indistinguishable from renal. Generally not crossed by bowel.

OVARIAN —Intestines behind. Grows from below upwards. Joined to the uterus. Loins are resonant. Urine is normal.

ENLARGED LYMPHATIC GLANDS.

FÆCAL ACCUMULATIONS.

MALIGNANT GROWTH OF LARGE INTESTINE.—Lacks shape of kidney. Symptoms referable to bowel. If large, is generally very fixed.

GROWTHS of mesentery, omentum, pancreas, gall-bladder.

PERINEPHRIC AND PARANEPHRIC TUMOURS —Bulge out into the loin. Mobility is very restricted Cannot be felt anteriorly.

MALIGNANT DISEASE OF KIDNEY

Aetiology.—Calculus is only a predisposing factor in carcinoma of renal pelvis.

Pathology.—

HYPERNEPHROMA (*see* p. 515).—Spheroidal and encapsuled. Metastases in lungs and bones. Proliferation of convoluted tubes. Spaces or tubes lined with renal epithelium. Probably a carcinoma of the renal tubules. Generally in adults, between forty and sixty.

CARCINOMA.—In old people, fifty to sixty-five Commoner on right side and in men. Two forms· capsulated and diffuse. Invades neighbouring viscera, especially colon or duodenum, and vena cava. Alveolar structure, with blood cysts. Metastases in lungs and liver.

SARCOMA.—In children under five, and less often in adults, twenty to sixty. Grows to enormous size. Invades vena cava, ureter, and lymph glands. Encapsuled in early stages. Hæmorrhagic and pseudo-cysts.

MYOSARCOMA occurs especially in infants. Probably congenital.

Symptoms.—

HÆMATURIA.—Occurs in 80 per cent. First symptom in over 50 per cent. Spontaneous. Uninfluenced by repose or exertion. Profuse generally. Often occurs at intervals and lasts six days. Sometimes clot-moulds of ureter. Not so common in children.

TUMOUR.—First symptom in 25 per cent adult cases. First symptom in majority of children Often difficult to detect, especially in upper pole Absence of tenderness usually. Jaundice, constipation, and vomiting sometimes occur when right kidney is affected.

PAIN.—Often absent in children. First symptom in 35 per cent adults. Begins in loin. Radiates to thorax, thighs, genitals. Uninfluenced by rest. Intermittent.

URINE.—Usually polyuria. Diminished urea and chlorides. Pus or albumin rare. May be frequent micturition.

VARICOCELE.—Comparatively sudden occurrence. Rapid development.

GENERAL SYMPTOMS.—Anæmia, emaciation, and 'cachexia'. Appear late (one or two years), and develop rapidly.

	Carcinoma and Hypernephroma	Sarcoma	
		Adult	*Child*
Age	40–60	20–40	5
Hæmaturia	Profuse in 70 %	Seldom profuse	Slight
Pain	Variable	Great	Slight
Cachexia	Early	Late	Early
Duration	3–3½ years	5–6 years	Less than 1 year
Metastasis	Usual	Rare	Rare

Diagnosis.—

1. WHERE TUMOUR IS PRESENT BUT NO HÆMATURIA:—
   Malignant disease is probable: In children under ten and adults between forty and seventy. Rapid growth. No periodic lessening in size. Venous obstruction—swelling of the leg or varicocele. Enlarged glands. Certain diagnosis can only be made by pyelography. Always exclude intrathoracic secondaries by radiography prior to operation.
2. WHERE HÆMATURIA IS PRESENT BUT NO TUMOUR:—
   a. Renal Origin.—Blood thoroughly mixed with urine. Passage of worm-like clots. Cystoscopic examination.
   b Nature of Renal Disease.—
   Calculus—Movement provokes hæmorrhage. Colic.
   Tubercle.—Hæmaturia slight. Pyuria. Bacilli can be found. Patient is a young adult.
   Nephritis.—Casts. Albumin in excess of blood.
3. WHERE TUMOUR AND HÆMATURIA CO-EXIST:—
   Diagnosis from tuberculous disease and calculous disease (*see* pp. 507, 510). Polycystic kidney—polyuria, anuria, or uræmia; generally double. Cysts. Hydronephrosis caused by tumour of bladder.

Treatment.—

NEPHRECTOMY.—25 per cent of cases die, and of the 75 per cent recoveries the majority have recurrence within three years

Transperitoneal for large growths. Lumbar for small.

A Combination of both incisions gives best results, because adhesions and pedicle can best be dealt with and drainage provided for.

Hypernephroma.—Hypernephroma or Grawitz's tumour, formerly believed to be a tumour arising in an adrenal rest, is probably a carcinoma arising from the renal tubules.

SITUATION.—It is usually connected with the kidney, developing as a nodule under the capsule, or as a distinct tumour in the kidney substance (*Fig* 180). More rarely it may be found in connexion with the ovary, testis, or uterus.

STRUCTURE.—It is a matter of dispute whether it is essentially an adenoma, carcinoma, or sarcoma. Probably it always begins as a benign adenoma, and subsequently becomes malignant. Shaw Dunn considers that it starts as a papillary adenoma. In its first stage it is definitely encapsuled; but later it breaks through the capsule, and invades the tissues round it, spreading along the veins and causing metastases. It is always of tubular structure, resembling the tissue of the adrenal cortex.

Hypernephroma—Structure, ***continued.***

The cells are markedly vacuolated, and contain notable quantities of fat, glycogen, and lecithin. The growth is very vascular, and occasionally contains giant cells.

SYMPTOMS.—These are usually those of a renal carcinoma, the following points being especially characteristic. Occur after middle age. There is often a long history of intermittent hæmaturia before the signs of a tumour occur. Later the hæmaturia becomes severe, and is accompanied by passage of clot-casts of the ureter and colic. Anæmia is well marked. Occasionally pigmentation of the skin occurs like that of Addison's

METASTASIS.—Occurs by means of the veins. The lungs, liver, or bones may be affected. Secondary growth in the bone may be the first evidence of the disease. The skull, humerus, and femur are those most often affected.

PROGNOSIS AND TREATMENT--Are similar to those of carcinoma of the kidney.

BENIGN TUMOURS OF KIDNEY

Adenoma.—Generally only size of pea and causes no symptoms. May have papillary and cystic formations.

Fibroma.—

Lipoma, Osteoma, and Chondroma.

Angioma, Lymphangioma, and Myoma.

Cysts of the Kidney.—(1) Retention cysts in granular kidney; (2) Dermoid cysts (very rare); (3) Simple cysts; (4) Conglomerate cysts (5) Hydatid cysts; (6) Paranephric cysts.

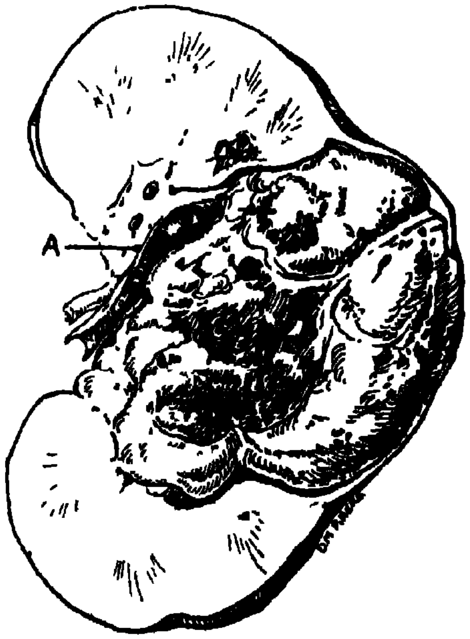


Fig. 180.—Section of kidney showing an early hypernephroma. A, Blood-clot in the ureter.

SIMPLE CYSTS.—Generally single, size varies from that of a cherry to a melon. At one pole of kidney. Filled with watery or colloid material. No symptoms except pressure. Treatment by partial nephrectomy.

POLYCYSTIC DISEASE OF THE KIDNEYS

Pathology.—Weight 1 to 16 lb. Retains kidney shape. Multitude of cysts from pin's head upwards. Lined by cubical epithelium. Contains fluid or colloid material. Almost always bilateral. Often associated with cystic disease of liver and spleen. Hypertrophy of left ventricle common. In infants it is associated with various congenital defects.

THEORIES OF ORIGIN.—Inclusion of mesonephric elements (Wolffian body). Failure of union between excretory tubes and ducts Feebleness of tubules, which renders them liable to dilatation. Cirrhosis or sclerosis, obstructing tubules. New growth, e.g., colloid adenoma.

Aetiology.—Either infants—'congenital cystic abscess'—or between ages of twenty and eighty. Males more often than females.

Symptoms.—

VERY INSIDIOUS ONSET, resembling chronic Bright's disease.

PAIN.—Dull aching in loins Sense of weight and discomfort. Some tenderness.

TUMOUR on one or both sides in about 50 per cent.

GENERAL SYMPTOMS.—Anuria or polyuria sometimes. Intermittent albuminuria or hæmaturia.

Urine resembles that of chronic Bright's—Pale, low specific gravity, few casts, variable albumin. Nausea, vomiting, flatulence, etc. Headache, delirium, convulsions, coma.

Cardiac and pulmonary symptoms, dyspnœa, bronchitis, etc.

Cachexia in only a few cases.

Diagnosis.—By pyelography Combinations of tumours with symptoms of chronic Bright's.

Prognosis.—Slow progress towards uræmia (one to ten years).

Treatment.—Nephrectomy if only one kidney be affected, and only if that is causing great pain or bleeding. Conservative operations (removal or puncture of cysts causing pressure on vessels or pelvis) are sometimes of great value in preventing progressive renal destruction.

TUMOURS OF RENAL PELVIS

Commonest Varieties.—(All potentially malignant.) (1) Papilloma; (2) Papillary carcinoma. (3) Spheroidal-celled carcinoma. (4) Squamous-celled carcinoma (metaplasia due to chronic infection or stone).

Piece of growth may become detached and grafted on ureter or bladder. Often cause hydro- or hæmatonephrosis. Vary from size of pea to huge mass. Often associated with and due to calculus.

Symptoms.—

TUMOUR is only palpable in advanced cases or where it has caused marked obstructive hydronephrosis. Sometimes tumour alternates with hæmaturia.

HÆMATURIA.—Earliest symptom. May be slight, intermittent, or fatal,

Tumours of Renal Pelvis—Symptoms, ***continued.***

URINE.—May have albumin or pus. Varies in amount.

PAIN.—Unusual. May be colic due to passage of clots. May be due to distension.

GENERAL.—Anæmia, etc. Uræmia (late).

Diagnosis.—Generally impossible from tumours of renal parenchyma. Presence of villous tufts in urine when cystoscope shows bladder normal.

Treatment.—Nephrectomy for squamous-celled carcinoma, as these tumours do not implant along the ureter. Nephro-ureterectomy essential in all papillary growths.

SUBCUTANEOUS INJURIES OF URETERS

Aetiology.—Result generally from crushes, kicks, or falls. Possibly from compression of ureter against transverse vertebral processes. Possibly from a violent falling forward of the kidney. Often in young people (below twenty-five).

Pathology.—Ureter torn right across or lacerated. Peritoneum generally not torn.

If peritoneum is torn, urine is extravasated into peritoneal cavity.

If the tear in the ureter is extraperitoneal, a urinary extravasation into loin occurs.

If the injury does not tear through, but obstructs ureter, some form of nephrectasis results.

Symptoms.—

HÆMATURIA.—Slight, severe, or absent.

URINE.—May be suppressed if both kidneys are injured.

PAIN.—As in kidney injuries.

TUMOUR.—If patient survives the accident, either an abdominal or loin tumour results.

If tumour is due to extravasation outside kidney, it appears in a few days.

If tumour is due to nephrectasis, it only develops in weeks.

Suppuration occurs in tumour generally.

Diagnosis.—Is only apparent after the formation of the tumour following an injury.

Prognosis.—Is bad only when associated with severe injury to both kidneys, or rupture of the peritoneum.

Treatment.—Puncture is unsatisfactory.

LUMBAR INCISION and drainage are the best routine treatment.

SUTURE URETER if rent can be found. Do not ligature proximal end of ureter.

NEPHRECTOMY should only be a last resort in case of severe suppuration or intractable fistula.

OPERATION WOUNDS OF URETER

Commonest in pelvic portion of the ureter. Near cervix uteri, or in the midst of adhesions or malignant growth. RESULT either in cutaneous or vaginal fistula, or peritonitis, or localized peritoneal urinary abscess. Injury is often partial and due to inclusion in stitch or ligature; urinary leak does not develop usually at once but often appears after a few days.

Treatment.—

1. URETERIC CATHETERIZATION may be successful in partial injuries. Tie in catheter for a few days.
2. RE-IMPLANTATION INTO BLADDER if injury is low down.
3. TRANSPLANTATION into bowel.
4. NEPHRECTOMY.
5. NEPHROSTOMY may be necessary when both ureters are severed—a life-saving measure.

End-to-end anastomosis of ureter is always followed by retrograde dilatation and is rarely satisfactory.

URETERITIS

Causes.—

1. INFECTION.—May be associated with any infection of the upper urinary tract and gives rise to dilatation and stasis Is of little clinical importance *per se* except in tuberculosis of the kidney, in which condition it may keep up infection after nephrectomy Hence the necessity of always removing the entire tuberculous ureter (nephro-ureterectomy) at the primary operation
2. CALCULUS.—Infection behind a calculus impacted in ureter gives rise to ureteritis resulting in dilated rigid ureter Such a ureter is unable to contract and the stone becomes 'silent'. Hence 'giant' ureteric calculi are usually symptomless.
3. EXTRINSIC CAUSES.—Involvement in fibrous tissue due to any severe pelvic inflammation.

Anatomy.—Three varieties:—

ACUTE.—Septic inflammation of mucous membrane. Little or no dilatation or induration.

CHRONIC DILATED FORM.—Ureter may be size of small intestine Great tortuosity at both ends Valves form at the bends. Canal is tortuous and obstructed. Little or no peri-ureteritis Great increase of fibrous tissue of ureter. Often cysts in mucous membrane (ureteritis cystica).

FIBROUS TYPE —Intimately adherent dense mass of peri-ureteral tissue. Ureter straight, thickened, and indurated All coats inflamed, with excess of fibrous tissue Strictures are long, rigid, and almost impermeable, or may be annular Often associated with single pyonephrosis.

Condition of Uretero-Vesical Orifice.—Generally contracted Sometimes dilated. Valve is destroyed by: (1) Mechanical effects of retention: (2) Inflammatory effects of cystitis.

OBSTRUCTION OF URETER BY VALVES OR STRICTURE

Causes of Valve Formation.—Temporary obstruction (e g., pregnancy, etc.) causes kinking and dilatation of renal pelvis. The pregnancy dilatation is not due to obstruction but is hormonal in origin. Inflammatory dilatation in uretero-pyelitis. Enfolding of mucous membrane round a stone. Actual valves in the ureter are rare.

Obstruction of Ureter by Valves or Stricture, ***continued.***

Causes of Stricture.—

CONGENITAL.—Generally close to renal pelvis. By aberrant blood-vessels.

ACQUIRED.—Injury. Cicatrices following ulceration. Impaction of calculus. Ureteritis.

Symptoms.—Commonest in females about thirty and in right ureter.

TUMOUR, either kidney or ureter

PAIN recurring at intervals of weeks Acute, resembling renal colic. Dull aching in loin in intervals.

URINE may contain Pus, or Crystals, or Blood.

TENDERNESS on bimanual examination, reduction of tumour, and filling of bladder.

BLADDER.—Frequency of micturition Inability to retain water. Pain on passing water.

STRICTURE can sometimes be felt per vaginam or felt with the ureteral catheter.

Treatment.—Exploratory nephrotomy. Pass bougie from above, if possible. Pass bougie from below.

1. If stricture or valve exist at junction of pelvis and ureter: Longitudinal incision. Sewn together as transverse incision
2. Nephro-ureterectomy or nephrectomy, where kidney is destroyed by dilatation (often necessary for 'giant' silent ureteric calculus

CALCULUS IN URETER

Aetiology.—In great majority of cases renal in origin. May form round foreign body, e.g., blood-clot. May remain *in situ* indefinitely, or be discharged within eighteen months, and kidney resume its functions.

SITUATION.—(1) Just below commencement of ureter; (2) At vesical orifice (especially in women); (3) At brim of pelvis.

Symptoms.—Colic. Pain shooting along ureter Tenderness over ureter. Hæmaturia. Anuria (if opposite kidney is blocked or diseased). Abdominal tumour (persistent or intermittent). Urine contains crystals, blood, or pus. Prolapse of ureter into bladder. Radiograph shows stone.

Diagnosis.—In the majority of cases the symptoms are simply those of renal calculus, and diagnosis is complete only on lumbar exploration and passing ureteral sound.

In some cases the stone can be felt per rectum or vaginam.

FROM CALCULUS IN A VESICAL DIVERTICULUM.—Impossible clinically.

FROM CYSTITIS.—In this: Urine is alkaline Pus at beginning or end of micturition.

FROM URETERITIS.—Difficult in absence of history of colic.

FROM VESICAL TUBERCULOSIS.—In this: Tubercle in urine. Polyuria. Frequent micturition. Slight hæmaturia. Symptoms not relieved by rest.

FROM PROLAPSED INFLAMED OVARY.—Ovary lies behind broad ligament and at greater distance from vaginal wall. Stone is felt in antero-lateral fornix. Hardness and outline are more definite.

RADIOGRAPHY shows a shadow in the course of the ureter, but this has to be distinguished from that of calcareous iliac glands or that of an appendix concretion (the latter very rarely throws a shadow).

INTRAVENOUS PYELOGRAPHY will show the ureter outline above the stone.

URETERAL CATHETERIZATION through the cystoscope not only shows which side is affected, but the end of the catheter may touch the stone and a waxed tip receives a scratched impression

Prognosis.—

KIDNEY.—Atrophy if block is complete and permanent. Recovery if block is removed within six to eighteen months. Hydronephrosis if block is incomplete. Pyonephrosis if sepsis occurs.

LIFE.—Not affected if other kidney is good. Endangered by inflammation and suppuration of kidney, extravasation of urine, anuria if opposite kidney is bad.

Pathological Effects.—

ON URETER.—

ABOVE.—Thinned, dilated, sacculated, and tortuous. Or œdematous, inflamed, and ulcerated, with rupture.

AT THE POINT OF IMPACTION —Ulceration and stricture.

BELOW.—Unchanged, or thickened and stenosed Prolapse of ureteral orifice. Ulceration of stone into bladder.

ON PERI-URETERAL TISSUE.—Induration, inflammation. Abscess or fistula.

ON KIDNEY.—Any variety of atrophy—Inflammation—Cystic kidney—Hydro- or pyonephrosis—Perinephritis and suppuration—Fistula.

Treatment.—Except when causing anuria (*see* p. 511), the case should be kept under observation in order to note whether the stone is fixed or moving. In the latter case time should be given for natural expulsion. To facilitate this, meatotomy of the ureteral orifice and dilatation of the ureter distal to the stone should be carried out. Injection of paraffin or oil into the ureter may be of value in some cases.

IMPACTED NEAR THE KIDNEY.—Attempt to push back and remove through the renal pelvis. Failing this, removal through an incision in the ureter, which is then sewn up.

IMPACTED AT THE BRIM OF THE PELVIS.—Remove through a long oblique lumbar incision which is produced down in front of the iliac spine.

IMPACTED AT THE ISCHIAL SPINE.—Distend the bladder and make a median sub-umbilical incision opening the cave of Retzius. Empty the bladder and separate it from the corresponding side of the pelvis, and work towards the spine of the ischium.

IMPACTED IN THE BLADDER WALL.—Remove through the bladder by a suprapubic operation. In women it may be done through the vagina, but this involves the risk of vaginal fistula.

RENAL INSUFFICIENCY

The incompetence of the kidneys to carry out their excretory functions. It may be absolute or only relative Normally there exists more than twice as much kidney substance as is necessary for the vital economy. If by atrophy, disease, or blocking of the ureters some of the kidney substance is destroyed or rendered functionless, a limit is reached beyond which any further kidney loss will be fatal. This is a matter of great importance in all operations upon the kidneys, ureters, prostate, or bladder, because either by direct removal of kidney substance or by severe shock the line between relative and absolute insufficiency may be overstepped.

Methods of Estimating Renal Insufficiency.—

THE AMOUNT OF THE URINE AND ITS CONSTITUENTS PASSED IN TWELVE HOURS.—

QUANTITY varies from 500 to 700 c c

UREA varies from 12 to 15 g

CHLORIDES vary from 5 to 6 g.

Any diminution of these quantities below 30 per cent indicates the lowest limit of renal sufficiency

INTRAVENOUS PYELOGRAPHY —The most reliable test for total renal function

UREA CONCENTRATION TEST —This is the most practical test, and the one used more than any other before deciding on operative treatment. All fluid is withheld for six hours A sample of urine is then collected. The patient is now given by mouth 15 g of urea in 100 c.c. water. Three specimens of urine are collected at hourly intervals and the urea in each is estimated If amount is over 2·5 per cent, kidney function is good. If not less than 2·0 per cent, kidney function is probably adequate.

INDIGOCARMINE TEST —This pigment may be given as an intramuscular or intravenous injection (4 c.c of a 0·4 per cent solution) The blue should appear within seven minutes if given intravenously, or within half an hour if given intramuscularly Its elimination ought to cease in twelve to twenty-four hours. Renal insufficiency is indicated by: (1) A late appearance of the blue in the urine; and (2) Prolonged elimination lasting more than three days.

CHROMOCYSTOSCOPY.—In normal cases, after indigocarmine injection the dye should be visible as a blue stream through both ureteric orifices within five to twenty minutes, being best seen thirty minutes later. The absence of one kidney or a blocked ureter is obviously demonstrated by this method.

PHENOLSULPHONEPHTHALEIN TEST.—An injection of 6 mg. of the dye is made, and the amount excreted within two hours estimated by colorimetric methods. The proportion of the original dose normally excreted in that time (60 per cent) is reduced when renal insufficiency exists.

ESTIMATION OF BLOOD-UREA.—

Blood normally contains from 10 to 40 mg. of urea per 100 c.c. of blood. It varies according to diet, fasting, and exercise.

It is tested by addition of the ferment urease (from Soya beans) to the blood, when the urea is split up, giving off nitrogen.

Any amount above 45 mg. per 100 c.c. indicates retention of urea, i.e., renal inefficiency.

In impending uræmia amount rises to 200 mg. or over.

Routine Method for Estimating Renal Insufficiency.—The functional capacity of one or both kidneys cannot be estimated by any one test. Clinical examination is at least as valuable as any chemical test. The following is a good routine method.

CLINICAL EXAMINATION.—

History of vomiting attacks; whether appetite, especially for meat, is good; presence of abnormal thirst

General appearance of patient; state of tongue.

Blood-pressure. State of arteries and heart.

Ordinary examination of urine. Large quantity and low specific gravity (i.e , below 1010) are signs of inefficiency.

CYSTOSCOPY AND UREA CONCENTRATION TEST —

If the cystoscopy is to be commenced, say, at 1.30 p.m., at 10.45 a.m. the bladder is emptied and a draught of 15 g. of urea in 100 c.c. of distilled water is swallowed

At 11.45 the patient passes urine= Specimen A.

At 12.45 the patient passes urine again = Specimen B.

At 1 30 the cystoscope is passed, under an anæsthetic if necessary, a catheter introduced into each renal pelvis, and 2 c.c of urine withdrawn from each kidney = Specimens C (r) and (l).

4 c c of a 0·4 per cent solution of indigocarmine is injected into a vein of the arm. The ureteric orifices are observed for the passage of the pigmented urine, and the time taken.

EXAMPLES ILLUSTRATING METHOD —

BOTH KIDNEYS HEALTHY.—

Clinical Examination.—No vomiting attacks, no abnormal thirst, and a good appetite for meat. General appearance good. Tongue moist. Blood-pressure low, no arteriosclerosis or enlargement of the heart. Urine examination normal

Cystoscopy, etc.—

Bladder and ureteric orifices normal.

Specimen A = 1·5 to 1·7 per cent of urea
Specimen B = 1·9 to 2·1 ,, ,,
Specimen C (r) = 2·0 to 2·2 ,, ,,
Specimen C (l) = 2·0 to 2·2 ,, ,,
Indigocarmine through each ureter in less than 7 minutes

ONE OR BOTH KIDNEYS UNHEALTHY.—

Clinical Examination.—History of vomiting attacks, thirst, no appetite for meat. Cachectic appearance. Tongue dry. Blood-pressure high; thickened arteries and hypertrophied heart.

Cystoscopy, etc —

Bladder or ureteric orifices may be abnormal.

Specimen A = 1·2 10 1·3 per cent of urea
Specimen B = 1·4 to 1·6 ,, ,,
Specimen C (r) = 1·4 to 1·5 ,, ,,
Specimen C (l) = 1·3 to 1·4 ,, ,,
No indigocarmine through either ureter in 10 minutes.

Estimation of Renal Insufficiency, ***continued.***

One Kidney Healthy, the other Diseased.—

Clinical Examination.—May reveal nothing abnormal.

Cystoscopy, etc.—

May show diseased condition of one ureteric orifice.
Specimen A = 1·5 to 1·7 per cent of urea
Specimen B = 1·9 to 2·0 ,, ,,
Specimen C (r) = 0·65 to 0·8 ,, ,,
Specimen C (l) = 2·5 to 3·0 ,, ,,

Indigocarmine through left ureter in 4 minutes, none through right ureter in 15 minutes.

Deduction is that right kidney is practically useless and can be removed with safety, as left kidney is 4 times as efficient as the right and is doing all the work.

Limitations of Renal Function Tests.—

1. Influenced by extra-renal factors (e.g., state of the heart, shock, hæmorrhage, etc.)
2. Tests only assess the state of the kidneys at the time of the test but do not indicate renal reserve.

Hence they must always be interpreted in the light of the clinical condition of the patient.

CHAPTER XLIV

AFFECTIONS OF THE BLADDER

CONGENITAL ABNORMALITIES

PATENCY OF URACHUS

There may be one of three conditions due to a persistence of the urachus connecting the bladder with the umbilicus.

Vesico-urachal Sinus.—A channel persists connecting the bladder with the umbilicus. Urine dribbles through this, especially if there is some obstruction to micturition, e.g., phimosis.

Umbilico-urachal Sinus.—A sinus leads down from the umbilicus to the patent urachus.

Urachal Cyst.—The urachus remains patent, but is closed above and below.

Treatment.—Of all varieties : excision.

ECTOPIA VESICÆ

Anatomy.—Bladder is only represented by the trigone bearing the ureteral orifices and part of the posterior wall. This part of the bladder is placed in the anterior abdominal wall in the hypogastrium The penis is rudimentary and shows complete epispadias. The pelvic bones are so ill-developed that the pubes do not meet at the symphysis. The pelvis is very shallow from before backwards. The umbilicus is not recognizable as such.

Symptoms.—Constant dribbling of urine. Excoriation of the skin. Eventual pyelonephritis. Metaplasia of exposed bladder mucosa leading to malignant change.

Treatment.—Intraperitoneal implantation of the ureters into the colon in all cases, followed by plastic operation for covering in the trigone.

RUPTURE OF THE BLADDER

Causes.—Direct blows on the distended bladder. By a wound from a portion of fractured pelvis. Penetrating wounds. Bursting following retention. Bladder is generally ulcerated or sacculated.

Varieties.—(1) Intraperitoneal; (2) Extraperitoneal.

Intraperitoneal Rupture.—Superior surface involved. Severe shock.
STRANGURY, i.e., frequent painful attempts at micturition.
SIGNS OF FREE FLUID in the abdomen.
PERITONITIS.

SIGNS.—**Catheter withdraws only scanty bloody urine., If lotion is injected into the bladder, less returns than is inserted.**

Intraperitoneal Rupture, ***continued.***

TREATMENT.—Laparotomy. Sponge out the free fluid. Sew up the bladder wound and drain suprapubically. Drain rectovesical pouch if the urine is septic.

Extraperitoneal Rupture.—Involves the base or lateral walls. Micturition may not at first be interfered with.

Pelvic Cellulitis or Extravasation of Urine occurs.

Dusky Swelling occurs above the pubes and in the perineum.

Rapidly Fatal Toxæmia if the urine is septic.

TREATMENT.—

1. Drain extra-vesical tissues wherever there is extravasation.
2. Drain bladder suprapubically, never by catheter.

CYSTITIS

Causes.—

ACUTE CYSTITIS —

1. Simple or catarrhal (*B coli*)
2. Hæmorrhagic due to staphylococcal infection.
3. Ulcerative due to mixed infection
4. Gangrenous (whole mucosa may slough) occurs in debilitated subjects suffering from long-standing obstruction (e.g , prostate or stricture); when it occurs it usually follows instrumentation

CHRONIC CYSTITIS.—Rarely occurs in a normal bladder. Should be regarded as a symptom and not a disease. Look for cause: Stone; Tumour; Large prostate; Stricture; Paralysis from spinal disease; Tuberculosis.

Bacteriology.—

Bacillus coli (alone)	In acid urine
B. tuberculosis „	
Gonococcus „	
Streptococcus „	
Bacillus proteus	Alkaline urine.
Diplococcus ureæ	
Staphylococcus	
Bacillus coli (with the above)	

Symptoms.—

PAIN in hypogastrium and perineum.

VESICAL IRRITABILITY.—Constant desire to micturate. Frequent micturition. Pain after micturition.

URINE.—

In Acute Cases.—May contain blood—Shreds and sloughs of the mucous membrane.

In Some Cases.—If infection is purely by an acid-forming organism (*B. coli*, gonococcus, tubercle bacillus), not much change is apparent. The organism can be recognized.

In Majority of Cases —Urine becomes alkaline by a formation of ammonia from the decomposition of urea. Foul smell. Loaded with pus, mucus, epithelium, and phosphates.

GENERAL SYMPTOMS (chiefly in the acute cases).—Toxæmia. Low febrile condition. Exhaustion from want of sleep.

Complications.—Septic infection of the kidneys.

Anatomy.—

ACUTE CHANGES.—

CATARRH —Acute inflammation of the mucous membrane.

MUCOUS MEMBRANE is intensely congested. Its vessels become thrombosed. Patches slough and leave ulcers. It may be cast off as a complete slough.

CHRONIC CHANGES.—

MUCOUS MEMBRANE, especially over the trigone.—Thickened and covered with tenacious mucopus mixed with phosphatic debris. Superficial layers lost. Congestion, with large dilated vessels. Sacculated and ulcerated.

MUSCULAR WALL.—At first thickened and hypertrophied. Later (if obstruction exists), becomes dilated, thinned, and fasciculated.

URETERS —Orifices are contracted by muscular spasm.

KIDNEYS.—Hydronephrosis. Pyonephrosis. Pyelonephritis.

Treatment.—

GENERAL.—In acute cases: Rest in bed. Milk diet.
Remove the cause (e g., stone, stricture).

DRUGS.—

COPIOUS ALKALINE DRINKS —To diminish irritating uric acid.

BELLADONA AND HYOSCYAMUS —For spasm

ANTISEPTICS —Hexamine, salol, benzoic acid, to prevent ammoniacal decomposition and to retard the growth of organisms. Mandelic acid. sulphonamides

DIURETICS —Citrate of potach, buchu, to diminish the concentration of the urine.

OLEO-RESINS.—Sandalwood oil, cubebs, copaiba, etc. In chronic cases. When there is much mucopus, and especially in chronic gonorrhœa.

TUMOURS OF THE BLADDER

Varieties.—

BENIGN.—Papilloma—Fibroma and myxoma (rare).

MALIGNANT.—Epithelioma—Columnar-celled carcinoma (secondary to uterine or rectal disease)—Sarcoma (rare).

PAPILLOMA OR BENIGN VILLOUS TUMOUR

In patients after middle age. Peak of incidence 60 years. Men more often than women.

Anatomy.—Tuft of branching processes. Each consists of central connective tissue and blood-vessels, clothed with transitional squamous epithelium. Submucous and muscular tissues are not involved. Generally at or near the trigone and close to one of the ureteric orifices. Occasionally multiple. May be secondary to villous tumous of the renal pelvis.

Symptoms.—Three stages: (1) Latent, (2) Hæmaturia, (3) Cystitis.

HÆMATURIA.—So-called symptomless hæmaturia, from the absence of other signs or symptoms. Profuse hæmorrhage, more marked at end of micturition. Recurs at long intervals, and lasts for a few days at a time.

Papilloma or Benign Villous Tumour—Symptoms, ***continued.***

VESICAL IRRITABILITY.—Frequency and pain rare. Pain may be due to difficult passage of clots.

CYSTITIS.—Rare in simple growths unless urine becomes infected.

HYDRONEPHROSIS (occasional), from infiltrating growths.

Signs.—Sound produces sharp hæmorrhage. Nothing felt by rectum or vagina. Cystoscope shows growth.

Prognosis.—There is often recurrence after operation, with ultimate malignant development.

Treatment.—

CYSTO-DIATHERMY OR FULGURATION.—An electrode from a diathermy apparatus is passed through a cystoscope and applied to the base of the papilloma, which is burnt off when the current is turned on. This method is very suitable for small growths.

OPERATIVE.—Suprapubic cystotomy. Isolate growth with a Fergusson's speculum. Press up its base from the rectum or vagina. Cut away the mucous and submucous tissue from which it grows with diathermy knife. Radon seeds may be implanted through operating cystoscope.

MALIGNANT NEW GROWTHS

May be primary, or secondary to growth of rectum or uterus.

Primary Growths.—Bladder growths pass insensibly from papilloma to papillary carcinoma to infiltrating carcinoma.

BRODER'S CLASSIFICATION:—

Grade 1 = Papilloma
Grade 4 = Infiltrating carcinoma } Grades 2 and 3 intermediate.

Symptoms.—Hæmaturia, dysuria, cystitis, pain in hypogastrium and perineum.

In malignant papilloma the hæmaturia precedes the dysuria, but in the other types dysuria precedes the hæmaturia by a considerable interval.

Complications.—Hydronephrosis with pain in the kidney from involvement of the ureter Pyelonephritis and septicæmia Retention of urine (rare), from involvement of the urethra.

Signs.—Indurated mass felt in the bladder wall, per rectum or per vaginam. Cystoscope shows a sessile papilloma or an ulcer with thickened margins. Sound may feel a rough area on the bladder wall.

Treatment.—

1. CYSTOSCOPIC IMPLANTATION OF RADON SEEDS into growth (small carcinoma unsuitable for removal—i.e., trigone).
2. PARTIAL CYSTECTOMY.—If the ureter is involved, the affected part is removed and the proximal end implanted into the bladder in a fresh situation.
3. TOTAL CYSTECTOMY.—In extensive growths. Ureters implanted in the colon or into the abdominal wall just internal to the anterior superior iliac spine.

DIAGNOSIS OF VESICAL GROWTHS

1. FROM OTHER CONDITIONS RESEMBLING THEM.—
   - *a.* **Renal Disease.**—Stone or new growth. In this: Pain or tumour in the loin. Hæmorrhage is much less profuse. Blood is evenly mixed with the urine. Segregation shows difference between the two sides. Urine remains acid. There is less vesical irritability.
   - *b.* **Stone in the Bladder.**—In this: Pain after micturition, with spasm, is present. Bleeding is slight. Sound feels the stone.
   - *c.* **Tubercle of the Bladder.**—In this: Pain and vesical irritability are severe. Bleeding is absent or trivial. Tubercle bacilli in the urine. Cystoscope shows shallow undermined ulcers
2. OF THE VARIETY OF GROWTH.—
   - **Benign Growths** are characterized by: Cystoscopic appearances. Absence of cystitis. Prompt response to diathermy (mobility with electrode). Profuse recurrent hæmaturia without other symptoms at first. Absence of induration of the bladder wall.
   - **Malignant Growths** are characterized by: Patient is over forty-five. Pain and cystitis are early and prominent Induration is felt in the vesical wall.

TUBERCULOUS DISEASE OF THE BLADDER

Tuberculosis of the bladder is almost always secondary to tuberculous disease of the kidney, prostate, or testicle.

Pathology.—Starts as a miliary tubercle in the submucous tissue. These caseate and break down, leaving ragged undermined ulcers. Generally situated in region of ureteric orifice of affected side.

Symptoms.—Those of chronic cystitis with hæmaturia. There is great irritability of the viscus.

Prognosis.—Unfavourable.

Treatment.—Deal if possible with primary focus. No local treatment. Bladder will improve with constitutional treatment after nephrectomy provided the disease is unilateral.

STONE IN THE BLADDER

Varieties.—

URIC ACID.—Oval or spherical stone. Brown in colour and laminated in texture (*Fig.* 181, *a*).

Deposits as rhomboidal, 'wheatsheaf', or irregular clumps of crystals.

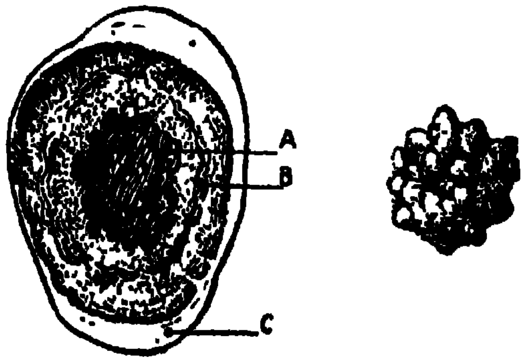


Fig. 181.—Vesical calculi. (*a*) Composite stone: A, Nucleus of oxalates; B, Layers of urates; C, Outer covering of phosphates. (*b*) Mulberry oxalate calculus. (× ½.)

Stone in the Bladder—Varieties, *continued.*

In acid urine, as result of excessive sweating, too little drinking, excess of nitrogenous food, sedentary habits, febrile condition, gouty diathesis. Much the commonest variety of stone.

AMMONIUM URATE.—Similar to the above, but lighter in colour. Deposits as spiculated globules or as an amorphous deposit.

CALCIUM OXALATE.—Rough, nodulated like a mulbery (*Fig.* 181, *b*). Very hard. Dark brown or blackish.
Deposits in acid urine, as 'envelope' or dumb-bell-shape crystals.

PHOSPHATIC CALCULUS.—Usually a secondary deposit on a uric acid or oxalate stone or foreign body. Sometimes primary in a saccule of a bladder, deposited by cystitis. Forms a white, soft, friable mass.
Deposits in alkaline urine as triple phosphate (ammonio-magnesium), 'knife-rest' crystals, or hexagonal feathery crystals. Calcium phosphate gives amorphous deposit. The mixed phosphates form a 'fusible calculus'.

CYSTIN.—Yellow, waxy—very rare.

XANTHIN.—Red—very rare.

Structure (*Fig.* 181, *a*).—

NUCLEUS at the centre formed by foreign body, renal calculus or mucus.

BODY.—Concentric layers of crystals held together by mucus.

CRUST.—Most recent deposit. Generally the mixed phosphates Occurs when cystitis with alkaline urine has been set up.

NUMBER.—Usually single Multiple where there is residual urine.

Aetiology.—

AGE.—Any age, but especially children under ten, men past middle life.

SEX.—Men much more often than women.

SOCIAL CONDITION.—In children it chiefly affects the poor.

CLIMATE, SOIL, etc —Very common in India and the tropics. Common in Eastern Counties.

LOCAL URINARY CONDITIONS.—Any obstruction predisposes to the deposit of a calculus: especially meatal stenosis in children, and a large prostate in old men.

Gout, lithiasis, or oxaluria usually precede and cause it, often first causing a renal calculus.

Symptoms.—

PREMONITORY.—Renal colic if stone originates in kidney. Incontinence of urine (in children). Passage of gravel, uric acid or oxalates.

PAIN.—In bladder and perineum. Referred to the end of the penis. Worse at the end of micturition.

FREQUENCY OF MICTURITION by day; by day and night when cystitis is present.

VESICAL IRRITABILITY.—Constant micturition, followed by pain and spasm, with sensation of not having emptied the bladder.

HÆMATURIA.—Slight and at the end of micturition.

INFLUENCE OF MOVEMENT.—All symptoms aggravated by movement, and therefore worse by day than night.

SUDDEN CESSATION OF MICTURITION or

ACTUAL RETENTION of urine, from impaction of the stone at orifice or in the urethra.

EFFECTS OF STRAINING and nerve irritation.—Piles or prolapse. Hernia. Priapism.

IN CHILDREN.—Incontinence of urine. Constant pulling at the foreskin. Symptoms masked by residual urine.

Signs.—

SOUNDING.—Bladder should be full of urine or lotion. Buttocks raised. Anæsthetic should be given in children. Beak is turned from side to side and then backwards behind a large prostate.

Hard click indicates a stone. It can be heard and felt. Most marked in oxalate or uric acid stones. Least marked in phosphatic stones. May be masked by mucus.

FALLACIES in sounding for a stone:—

Stone may be missed because: It lies deep behind a large prostate, or in a saccule with a narrow mouth, or it is covered with tenacious mucus.

Click or grating is given by conditions other than a stone, viz.: Prostatic calculus. Phosphatic debris on an ulcer or new growth.

RADIOGRAPHY.—All the common varieties of stones are well shown by the X rays

CYSTOSCOPY will reveal the actual stone.

Diagnosis.—From renal calculus, vesical new growth (*see* p. 529), vesical tuberculosis.

INCONTINENCE IN CHILDREN —Stone should always be suggested by marked priapism or intractability of the symptoms.

Complications.—

1. INFECTIVE COMPLICATIONS.—Cystitis, pyelitis, and pyelo-nephritis.
2. OBSTRUCTIVE COMPLICATIONS. — Hypertrophy and sacculation of bladder. Formation of diverticula. Hydronephrosis.
3. ULCERATION and CARCINOMA.

Treatment.—

PRELIMINARY FACTS to be ascertained:—

1. Presence of any urinary obstruction. Size and position of stricture. Presence and character of prostatic growth.
2. Size of the stone. By palpation through the rectum. By grasping with a lithotrite.
3. Nature and consistency of the stone. By examination of the crystals in the urine. By the smooth, or rough, or soft sensation imparted to a sound.
4. Presence of cystitis or kidney disease.

LITHOLAPAXY.—

INDICATIONS.—Stone less than 1½ in. in diameter. Absence of stricture or very large prostate or diverticulum. Absence of cystitis or kidney complications.

METHOD.—Buttocks raised. Bladder filled with boracic lotion (at least 6 oz.). Grasp the stone and crush it by lithotrite. Crush all fragments that can be grasped. Wash out all fragments by the evacuator. Use largest evacuating tube possible. Cystoscope, and treat any residual fragments.

Stone in the Bladder—Treatment, ***continued.***

SUPRAPUBIC CYSTOTOMY.—

INDICATIONS.—Large stone (1½ in. or more). Lithotrite can deal with any stone except when formed around a foreign body. Combined with a large prostate. When a stricture exists. When the stone is encysted.

METHOD.—Trendelenburg position. Fill the bladder until it is felt above the pubes. Open immediately above the pubes. After removal of the stone: always drain the bladder for a few days.

DIVERTICULUM OF THE BLADDER

Definition.—A sac-like protrusion of some part of the bladder wall.

Varieties.—

1. CONGENITAL.—These consist of a protrusion of the muscular coat. Rare, and exceedingly rare in females.
2. ACQUIRED.—These consist of a protrusion of mucous membrane only, and may be:—
   - *a.* PRESSURE DIVERTICULA.—These follow the sacculation produced by bladder-neck obstruction. Take many years to develop.
   - *b.* TRACTION DIVERTICULA.—These are produced by traction from without—e.g., hernia, perivesical inflammation.

Symptoms.—Severe and intractable cystitis from urine stagnating in the diverticulum. May give rise to an encysted calculus, which may ulcerate through the diverticulum, producing extravasation of urine.

Diagnosis.—Made by X rays after filling bladder with sodium bromide; and by cystoscopy, when the opening of the diverticulum appears as a black aperture in the bladder wall.

Treatment.—Excision of the diverticulum and removal of the cause.

INCONTINENCE OF URINE

Definition.—Involuntary micturition due to abnormal detrusor action or defective sphincter action.

1. Active Incontinence.—Abnormal detrusor action, common in children, especially boys. Generally at night—'nocturnal incontinence'.

CAUSES.—Reflex stimulation of abnormally excitable vesical centre. For example, by: Slight vesical distension—Rectal parasites or polypus—Phimosis—Uric acid gravel—Emotions.

TREATMENT.—Remove any abnormal cause of irritation.

Wake child to pass water before bladder is too full.

Bland diet: No meat in evenings.

Drugs: Iron and arsenic as tonics. Belladonna and bromides as sedatives.

2. Passive Incontinence.—Defective sphincter action.

a. PARALYTIC.—From injury of vesical centre in the cord. Sphincter is paralysed and remains relaxed. Urine dribbles out from the ureters to the urethra. Bladder remains empty and contracted.

b. MECHANICAL.—When a stone or growth holds open the neck of the bladder (very rare). Over-distension of female urethra.

c. TRAUMATIC.—Damage to both sphincters at operation (e.g., perineal prostatectomy). After difficult labour. (Mild degree—stress incontinence of Bonney.)

Treatment of Passive Incontinence.—That of its cause. Also an attempt to prevent cystitis by aseptic precautions at the external meatus.

3. False Incontinence.—Distension with overflow. Bladder is greatly distended, and the urine constantly dribbles away.

a. OBSTRUCTION to the outflow of urine. Enlarged prostate. Stricture of the urethra.

b. SPINAL CORD INJURY.

Treatment of False Incontinence.—That of the cause, with aseptic precautions.

RETENTION OF URINE

Causes.—

1. OBSTRUCTION TO THE OUTFLOW.—
   - In Penis.—Phimosis—Rings, bands round penis—Malignant growth (very rarely causes obstruction).
   - In Urethra.—Stricture—Impacted stone—Rupture.
   - In Prostate.—Hypertrophy—New growth—Inflammation.
   - In Bladder.—New growth—Stone (rarely causes retention).
   - Outside Neck of the Bladder.—Pressure of tumours, e.g., uterine fibroids—Retroverted gravid uterus.
2. NERVOUS.—Diseases of the spinal cord, especially tabes or myelitis. Injury of brain or spinal cord Injury of the reflex nerve mechanism, e.g., by operations on the rectum or genital organs. Hysteria.
3. ATONY of the bladder produced by: Over-distension—acute or chronic. Cystitis (rare). In old age (probably associated with prostatic enlargement).

Symptoms.—Bladder presents as a tense hypogastric swelling. Pain of a constant character in abdomen and perineum (this is absent in paralytic cases)

Results.—

CLINICAL.—(1) Rupture of the bladder; or (2) Rupture of the urethra; or (3) Distension with overflow—false incontinence.

PATHOLOGICAL.—

On the Bladder.—In acute cases: Dilatation and atony. In chronic cases: Hypertrophy, followed by dilatation and sacculation.

On the Kidneys.—Hydronephrosis or pyonephrosis. Pyelonephritis.

Diagnosis.—

SUPPRESSION OF URINE.—In this: The bladder is empty and contracted. Follows some severe shock, or operation, or nephritis. Scanty urine is of high specific gravity and albuminous.

CALCULOUS ANURIA.—History of lumbar pain and colic. Bladder is empty. Pain is absent or exists only in the loins.

RUPTURED BLADDER.—Follows an injury. Bladder is empty or contains bloody urine. More lotion can be put in than got out of the bladder.

Retention of Urine, ***continued.***

Treatment.—

1. WHEN CONGESTIVE SPASM IS THE CAUSE: First try hot baths, with morphia and belladonna suppositories. Then pass a soft catheter.
2. WHEN DEFINITE OBSTRUCTION EXISTS: Pass large soft catheter first: if this fails, then small or filiform catheters.
3. WHEN THE PROSTATE IS ENLARGED: First try large soft catheter, then catheter coudé, then metal prostatic catheter.
4. WHEN NO KIND OF CATHETER CAN BE PASSED:—
   Suprapubic Drainage.—Pass a de Pezzer's tube through the cannula and leave it in the bladder.
   Suprapubic Cystotomy.—When a large prostate or vesical growth can be dealt with later, or calculus at the same time.
   Perineal Cystotomy.—When a urethral stricture exists which can be treated at the same time.
   In acute retention of many hours' standing, and *in chronic retention with cystitis,* it is better to relieve the retention first by a catheter or aspiration, and do a radical operation later when the patient and the bladder are in better condition.

CHAPTER XLV

DISEASES OF THE PROSTATE

Anatomy.—Shaped like a pyramid, with four triangular surfaces. Normally measures a little over an inch transversely, about one inch in other diameters.

SURFACES.—

SUPERIOR SURFACE is in the cavity of the bladder. Is pierced by the internal meatus. Is covered by vesical mucous membrane.

POSTERIOR SURFACE is related to vesiculæ seminales. Separated from rectum by fascia and cellular tissue, and is covered by rectovesical fascia.

LATERAL SURFACES.—Lie on the origin of the levatores ani from the pubes. Are covered by visceral layer of pelvic fascia. This is connected to the pubis in front as the puboprostatic ligament.

APEX.—Situated on the junction between the lateral surfaces. Is pierced by the urethra Rests on that part of the pelvic fascia which forms the deep layer of the triangular ligament

CONNEXIONS —

BLADDER.—The mucous membrane is continued over its superior surface into the urethra. The muscular wall is continued into its posterior and lateral surfaces

URETHRA traverses the gland from its superior surface to its apex.

EJACULATORY DUCTS —Traverse its substance from its posterior surface to join the urethra

SURROUNDINGS.—The gland with its fibromuscular capsule is surrounded by.—

1. The neck of the bladder, which is structurally continuous with the fibromuscular capsule.
2. Cellular tissue containing the prostatic plexus of veins.
3. A sheath of fascia: (*a*) Deep layer of triangular ligament; (*b*) Visceral layer of pelvic fascia at the sides; (*c*) Rectovesical fascia behind. (These are all parts of the same fascia, forming a continuous 'SHEATH' for the prostate)

STRUCTURE. Consists of two kinds of tissue: (1) Mass of unstriped muscle and fibrous tissue; (2) Glandular tissue embedded in (1).

CAPSULE consists of the peripheral parts of the fibromuscular tissue arranged in a laminated manner. This dips down as a median raphe, and joins the anterior and posterior walls of the urethra. There is no distinct demarcation between the capsule and the rest of the gland in normal conditions.

LOBES.—Two primary lateral lobes lie on each side of the median raphe. A central, median, or third lobe is that part of the gland superior and posterior to the internal urethral meatus; it is divided from the lateral lobes by the ejaculatory ducts.

Diseases of the Prostate—Anatomy, ***continued.***

DUCTS.—The glands of the lateral lobes open by ten to fifteen ducts into the groove on the floor of the urethra at the side of the crista urethræ. The glands of the middle lobe open into a recess—the sinus pocularis—which opens on the summit of the crista urethræ. The ejaculatory ducts open on either side of the orifice of the sinus pocularis. The median lobe of the prostate is the uterus masculinus.

INFLAMMATION

Varieties.—Acute prostatitis, acute abscess. Chronic prostatitis. Tuberculous prostatitis.

Acute Prostatitis.—

CAUSES.—Gonorrhœa—Sepsis secondary to instrumentation—Stricture—Cystitis.

ANATOMY.—Inflammatory swelling of the whole organ. Ducts and follicles plugged by mucus and bacteria. Abscess forms: (*a*) In one of the follicles; or (*b*) In the parenchyma spreading through the capsule.

SYMPTOMS.—Burning pain in the perineum, worse on sitting. Frequent painful micturition. Retention of urine from spasm and congestion. Constitutional symptoms of fever. Constipation. Pain during and after defæcation

SIGNS.—Passage of prostatic shreds or pus in urine. Inflamed prostate felt per rectum. Brawny swelling in the perineum. When suppuration occurs under or outside the capsule, fluctuation felt by rectum or in the perineum.

COMPLICATIONS.—Abscess may burst into: Urethra—Bladder—Rectum—Perineum. Rectal or perineal fistula may result. Cystitis and septic disease of the kidneys

TREATMENT.—Rest and milk diet. Hot hip-baths. Fomentations to perineum. Hot enemata. Morphia and belladonna suppositories. For retention: Soft catheter. For suppuration: Median perineal incision.

Chronic Prostatitis.—

CAUSES.—Posterior urethritis (gonorrhœa)—Cystitis—Stricture.

SYMPTOMS.—Frequent micturition. Slight pain after micturition. Sense of weight in the perineum Discharge of albuminous material after micturition, especially the first thing in the morning. Passage of mucous casts of the prostatic glands. Rarely a chronic abscess forms.

SIGNS.—Tender enlargement felt per rectum.

COMPLICATION.—Formation of prostatic calculi.

TREATMENT.—Bland diet. Absence of sexual excitement. Copious fluids, with diuretics. Sulphonamides. Local treatment harmful unless calculi or marked fibrosis present; then dilatation is required to relieve the obstruction.

Tuberculous Prostatitis.—Usually secondary to tuberculous disease of testis or vesiculæ seminales. Rarely primary.

ANATOMY.—Caseous deposits in the prostate. Chronic abscesses. Ragged ulceration, generally the result of septic infection.

SYMPTOMS.—Those of chronic prostatitis. Pyuria, with urine which is acid at first and may contain tubercle bacilli. Rectal examination reveals a tender irregular enlargement of the prostate.

COMPLICATIONS.—In all except the mild primary cases it spreads to the bladder and kidneys.

TREATMENT.—Generally only constitutional. Bland diet with copious fluids. Morphia and belladonna suppositories for pain. Perineal aspiration for tuberculous prostatic abscess.

Prostatic Calculi result from deposit of lime salts in the follicles. In cases of chronic prostatitis. Consist of carbonate of lime. Usually multiple. Collect in enlarged prostatic pouches.

SYMPTOMS.—Irritability of the bladder Difficulty in micturition.

SIGNS.—Those of chronic prostatitis. Felt by a sound just before entering the bladder.

TREATMENT.—Regular dilatation Never operate unless calculi cause true obstruction with retention of urine. Under such circumstances the calculi can be removed through a median perineal incision

ENLARGEMENT OF THE PROSTATE

Aetiology.—Common after fifty. May be caused by prostatitis, urethritis, or lithiasis, or any condition of chronic vesical irritability. Usually is senile.

Morbid Anatomy (*Fig.* 182).—

PROSTATIC CHANGES.—

SHAPE AND SIZE —At first it retains its shape, then it becomes lobulated and irregular The lateral lobes enlarge towards the rectum.

The median lobe and the whole of the superior surface grow up into the bladder

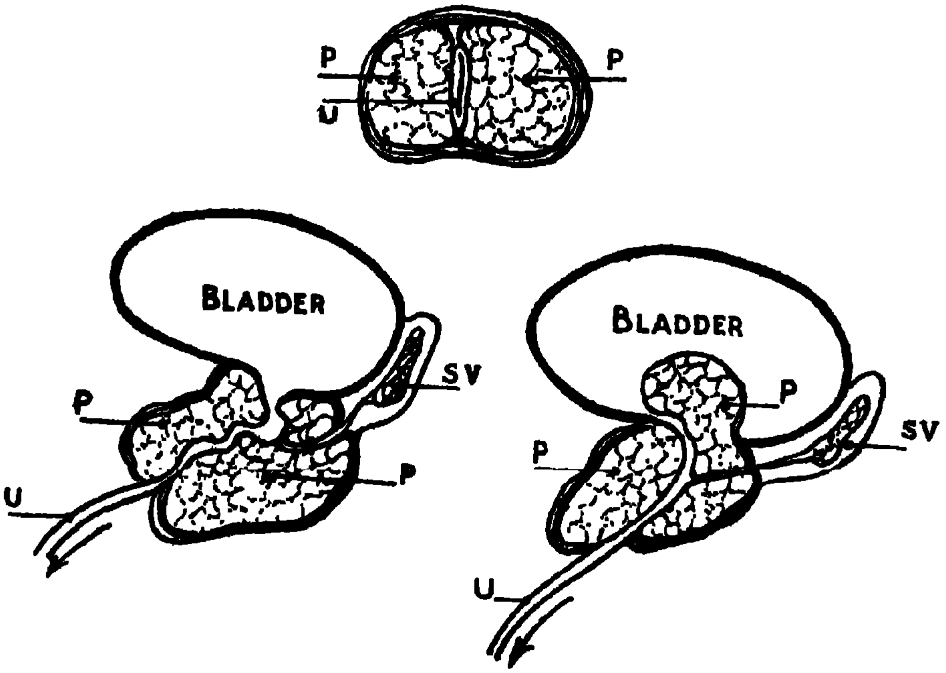


Fig. 182.—Diagram of prostatic enlargement. Upper figure is a transverse section of prostate, showing urethra (U) compressed by the lateral lobes of the prostate (P). The two lower figures are antero-posterior sections. That on the left shows an elongated and tortuous urethra; that on the right an elongated and curved urethra, obstructed by an overhanging middle lobe. S V, Seminal vesicles.

Enlargement of the Prostate—Morbid Anatomy, ***continued.***

A median constriction marks the attachment of the neck of the bladder and separates the intravesical from the extravesical parts of the gland.

STRUCTURE —The enlarged gland consists of either: (1) Excess of fibrous tissue: usual in the smallest specimens which cause symptoms. (2) Overgrowth of all the normal tissues: usual in the smooth, medium-sized specimens. (3) Fibro-myomatous tumours or fibroids: may form large masses. (4) Fibro-adenomata, i.e., overgrowth of the glandular tissue: constitute the bulk of the largest specimens. (5) A well-marked laminated capsule consisting of layers of fibro-muscular tissue surrounds the rest of the mass, and is continuous with the muscular walls of the bladder.

CONSISTENCY —Hard when chiefly fibrous tissue. Soft when formed by adenomata.

CHANGES IN THE URETHRA.—Elongated from one to several inches when intravesical growth occurs. Curved with the convexity backwards. Compressed from side to side Tortuous from the pressure of enlarged lobules Internal meatus may be distorted or blocked by the growth of the median lobe, or rarely held open by the same.

CHANGES IN THE BLADDER AND KIDNEYS.—The bladder becomes first hypertrophied and then dilated, the muscular fasciculi being separated, and the mucous membrane pouched between the fasciculi. Residual urine up to several ounces may accumulate in the retroprostatic pouch. Cystitis occurs sooner or later. Hydronephrosis, pyonephrosis, or pyelonephritis may result from chronic retention and cystitis.

Symptoms.—There are usually four stages, though the earlier ones may not be noticed or complained of, viz. :—

1. DIFFICULTY IN MICTURITION.—A long time is taken in starting the stream The stream is of good bulk but slow, and projected with little or no force. Straining often checks the stream.
2. FREQUENT MICTURITION —Due at first to congestion and irritability of the neck of the bladder, later to retention. Worse at night, or most noticeable then.
3. RETENTION OF URINE —The bladder cannot empty itself when the obstruction increases or when the bladder becomes weak. Hence some residual urine is left after each act. This varies from one to ten ounces, and constitutes the measure of the inefficiency of the bladder. Acute retention may be the first sign, or more commonly supervenes on chronic retention when micturition has been delayed so long that the bladder becomes atonic, or results from congestive spasm caused by cold or alcoholic excess. False incontinence (retention with overflow) may supervene in any neglected case
4. CHRONIC CYSTITIS.—Infection is usually by instruments. May arise by auto-infection. Urine becomes ammoniacal and loaded with phosphates, pus, and mucus.

Physical Signs.—

SIGNS OF RETENTION OF URINE in the absence of stricture or paralysis. Bladder felt above the pubes (rare in early cases). Presence of residual urine after micturition.

RECTAL EXAMINATION.—Gland is felt on the anterior rectal wall, about 2½ in. from the anus. It feels large or lobulated. It may feel practically normal.

URETHRAL EXAMINATION.—The prostatic urethra is elongated—judged from the distance the catheter must be passed from the pubic arch till the bladder is entered. Normally this is about 1½ in.; in prostatic cases it is anything up to 4 in.

A long curved catheter or a coudé passes most easily.

CYSTOSCOPE.—The intravesical enlargement may be seen with difficulty.

Treatment.—

1. DIET AND DRUGS.—For mild cases without any residual urine.

DIET.—Meat and rich food to be taken very sparingly Plenty of milk, alkaline waters, and diluents. Avoid alcohol.

REGULATION OF THE BLADDER.—Never postpone micturition after the desire occurs.

DRUGS —Alkalis, e g , bicarbonate and citrate of potash. Sedatives, e.g., hyoscyamus or belladonna. Antiseptics: hexamine, salol, to prevent or minimize cystitis

2. CATHETER TREATMENT.—For cases in which retention occurs, either acutely or chronically, shown by the existence of residual urine. It must be remembered that catheterization is only a temporary measure: (1) To relieve acute retention; (2) To decompress the bladder in chronic retention 'Catheter life' is a brief one owing to dangers of infection and hæmorrhage.

CHOICE OF INSTRUMENT —(*a*) No 12 rubber catheter; (*b*) Coudé or bi-coudé; (*c*) Metal catheter with long prostatic curve A rubber or metal instrument is better for regular use because it can be boiled.

METHOD —Passed once a day—preferably at night. Antiseptic precautions for the meatus and catheter. Efficient lubrication.

CONTRA-INDICATIONS —Existence of a tight urethral stricture. Great pain on catheterization, due to nervousness or irritability of the bladder. Bleeding on catheterization. Patients who cannot carry out the necessary antiseptic precautions. Patients who cannot pass the catheter themselves. Occurrence of rigors and fever (catheter fever). Cystitis (if this can be cured by irrigation of the bladder, the catheter may be resumed, but cystitis quickly recurs).

3. RADICAL OPERATIVE TREATMENT—PROSTATECTOMY —

INDICATIONS.—Increasing residual urine. Attacks of acute retention. Complications such as stone. infections. etc. Provided that the general condition of the patient warrants the operation.

a. SUPRAPUBIC OPERATION (Freyer's).—Fill the bladder with lotion. Open above the pubes. Incise the mucous membrane over the prostate. Press up the prostate by left fingers in the rectum. Insert the index finger between the bladder wall and the prostate through the opening in the mucous membrane. Sweep round the prostate between the layers of the capsule. Tear through the urethra as it enters the deep layer of the triangular ligament. Remove the prostate. Irrigate the bladder with hot water. Tie in a large drainage tube. Sew the bladder and parietes round the tube. Arrange some suction or siphonage

Enlargement of the Prostate—Treatment, ***continued.***

apparatus, e.g., Cathcart's irrigator or Irving's dressing. Patient should pass water by penis in ten to twenty days. Lessen the drainage gradually.

The *advantage* of this operation is speed. The *disadvantages* are: lack of control of hæmorrhage; slow healing; obstruction more common from flap formation from tags around the prostatic cavity.

b. TWO-STAGE OPERATION.—The two-stage operation is indicated where preliminary drainage of the bladder is advisable (e.g., sepsis, renal failure). It should always be done in the following conditions: retention severe enough to demand operative relief; cystitis; calculus; renal efficiency below or on the border line of safety. In the first stage the bladder is drained by a simple suprapubic cystotomy or by the insertion of a de Pezzer catheter through an abdominal stab incision. Drainage may be necessary for many months before the patient is fit enough for the second stage—namely, removal of the prostate.

Results.—(*a*) Complete recovery, with normal micturition in about 70 to 80 per cent. (*b*) Death in about 20 per cent of the cases from: Shock and hæmorrhage—Mania—Septic infection, especially through the kidneys—Senile asthenia—Uræmia. (*c*) Recovery with permanent suprapubic fistula. (*d*) Recovery with an atonic bladder which requires occasional catheterization.

c. OPEN SUPRAPUBIC, THOMSON-WALKER, OR HARRIS'S OPERATION.—A 4-in. incision is made and the bladder freely opened so that the hand may be inserted. The prostate is then enucleated without inserting the left finger into the rectum. The patient is now placed in the full Trendelenburg position and a self-retaining retractor inserted into the bladder. Any loose tags of prostatic tissue are then removed under direct vision and the bleeding points underrun and tied. The mucous membrane at the orifice of the cavity is trimmed, and a wedge is cut out of the prominent semilunar ledge forming the posterior lip of the opening. In Harris's operation the mucous membrane of the bladder is sewn to the cut urethra, so as to leave no raw surface. The bladder is closed without drainage. Original Harris operation with closure now rarely performed, but modifications thereof have many advantages over the older Freyer operation.

Advantages.—There is less risk of infection because there has been no trauma to the rectum. The risk of hæmorrhage is less, and owing to cutting away the semilunar ledge there is very little possibility of post-operative fibrous narrowing of the vesico-prostatic opening.

d. TRANSURETHRAL RESECTION.—

Indications.—Cases of moderate enlargement, but marked obstruction.

Method.—The instrument (resectoscope) is a kind of cystoscope with a wire snare, connected to a diathermy current. Portions of the gland abutting on the urethra are removed until the passage is sufficiently enlarged.

e. PERINEAL PROSTATECTOMY.—Very low mortality but operation requires special training and technique. Functional results not so good as suprapubic method.

All operations for removal of the prostate should be preceded by vasotomy.

MALIGNANT DISEASE OF THE PROSTATE

This is always a round-celled carcinoma. Its ordinary symptoms and signs are indistinguishable from those of benign prostatic enlargement, and in its early stages it is seldom recognized as malignant before operation. It may be symptomless, first manifestation being a secondary deposit giving rise to sciatica, œdema of legs, constipation due to occlusion of rectum, spontaneous fractures. The following points may indicate its nature :—

1. The rapidity of development of the symptoms of urinary obstruction.
2. Pain and cystitis will be earlier and more conspicuous than in simple enlargement.
3. By the rectum the growth feels large, hard, and nodular, and later the rectal walls become fixed to it.
4. The mucous membrane is fixed and not freely movable over the prostate in late stage.
5. The median raphe is soon lost
6. Serum acid phosphatase is increased when growth has metastasized.

TREATMENT.—If its nature is not recognized before suprapubic enucleation is performed, the operation may prove of exceptional difficulty, the growth tearing instead of shelling out Bleeding will be unusually profuse and recurrence early and rapid.

PERINEAL PROSTATECTOMY should be performed if malignancy is recognized before operation. After exposure of the gland and division of the membranous urethra, the prostate is removed, together with its proper capsule, from inside its sheath of pelvic fascia. The bladder walls are cut at their junction with the prostate, and the latter is removed. The bladder is sewn to the urethra over a rubber catheter.

RADIUM.—A suprapubic cystotomy is performed, and radon seeds or radium needles are buried in the prostate, but the results are poor.

ENDOSCOPIC RESECTION will often relieve the obstruction.

STILBŒSTROL (1 mg. t.d.s.) causes diminution in size of tumour, delays spread of metastases, and gives rapid and dramatic symptomatic relief.

CHAPTER XLVI

AFFECTIONS OF THE URETHRA

TRAUMATIC RUPTURE

Causes.—Falling astride. Blows on the perineum. Fracture of the pelvis.

Situation.—Membranous urethra, usually as it passes through the (superficial) triangular ligament.

Extent.—Partial, through the floor; or entire, through the whole circumference.

Symptoms.—Pain. Retention of urine.

Signs.—Ecchymosis and swelling of perineum, scrotum, and penis. Extravasation of urine if micturition is attempted. Bleeding from the urethra.

Result.—Fibrous stricture of a dense resilient character, or death from extravasation.

Treatment.—*In all cases, immediate suprapubic cystotomy.*

IF NO PERINEAL BLEEDING OR SWELLING OCCURS: Adopt no local treatment. Rest in bed

IF PERINEAL SWELLING OCCURS: Open the urethra at once. Find the ends of the torn portion. Sew together over a soft catheter. Leave catheter in for one week.

IF EXTRAVASATION OF URINE HAS OCCURRED: Open the urethra in the perineum; pass a tube into the bladder. Make multiple incisions over the swollen parts. Immediate union of the ends of the urethra over the catheter in recent cases.

STRICTURE OF THE URETHRA

Anatomy of Normal Urethra.—

1. PROSTATIC URETHRA.—Widest and most dilatable part of urethra.

   LENGTH.—One inch or more

   DIAMETER —Large, admitting No 20 sound. About ¼ in.

   SHAPE.—On section, horseshoe-shaped with convexity (verumontanum) forwards.

   STRUCTURE.—

   Median ridge—the crista urethræ—runs along the floor On this opens mesially the sinus pocularis, and at the sides the ejaculatory ducts.

   Lateral grooves into which the ducts of the lateral lobes of the prostate open.

   DIRECTION.—Vertical.

   STRICTURE.—Very rare; only as result of trauma or operation such as prostatectomy.

2. MEMBRANOUS URETHRA.—

LENGTH.—Half an inch.

DIAMETER.—About ¼ in. Admits a No. 12 sound with some tension. Narrows in its passage through the superficial triangular ligament.

POSITION.—Runs from the prostatic sheath, i e., the deep triangular ligament, to the superficial triangular ligament.

SURROUNDED by the compressor urethræ. Cowper's glands lie on each side.

DIRECTION.—Downward and forward

STRICTURE.—Always traumatic.

3. SPONGY OR PENILE URETHRA.—

LENGTH.—Six inches or more.

DIAMETER.—¼ in. No 12 sound fills it without tension. Narrowest point of whole canal is at the external meatus.

SHAPE.—Horizontal slit, except at meatus, where it is a vertical slit.

STRUCTURE.—

Mucous membrane is lined with columnar cells Lacunæ open into it. The lacuna magna is larger than the rest, and opens into the upper wall far back near the beginning. Minute simple glands —glands of Littré—also open into it.

Very vascular submucous layer underlies the mucous membrane.

Unstriped muscle is round this, and

Erectile tissue of corpus spongiosum surrounds the whole.

DIRECTION.—First horizontal Then upwards and forwards. Then in the line of the penis.

STRICTURE.—Inflammatory.

Varieties of Stricture.—(1) SPASMODIC AND CONGESTIVE; (2) INFLAMMATORY, (3) TRAUMATIC.

Spasmodic and Congestive Strictures.—

SITUATION.—In the bulbous urethra.

CAUSES.—Acute urethritis. Slight blows on the perineum. Cold and wet. Alcoholic or sexual excess, especially when some organic stricture co-exists. Injury from passage of instruments. Operations on the rectum, perineum, or testis. Parasympathetic stimulants.

TREATMENT.—Hot bath. Hot enema. Morphia and belladona suppository If retention is unrelieved, large soft catheter.

Inflammatory Strictures.—

AGE.—Commonest in patients from twenty-five to fifty.

CAUSES.—Gonorrhœa—by far the commonest. Ulcer caused by a foreign body. Urethral chancre—very rare.

PATHOLOGY—The mucous membrane is subject to a suppurative catarrh. At certain spots this penetrates deeply, probably where glands or lacunæ are involved. The mucous membrane here is destroyed and an ulcer produced. The organisms and inflammatory process are thus admitted to the vascular submucous tissue In the submucous tissue a chronic plastic inflammation occurs, producing a round-celled infiltration. This in time is converted into scar tissue, the contraction of which causes the stricture.

THE STRICTURE.—Mucous membrane is lost by ulceration. Mass of cicatricial fibrous tissue occupies the rest of the urethral wall and

Inflammatory Strictures—Pathology, *continued.*

penetrates into the spongy body to a greater or less extent. Malignant disease is a rare complication which may be superadded to stricture. It is usually a transitional-celled carcinoma.

Urethra Above the Stricture.—Dilated. Septic inflammation, with the loss of the superficial layers of the mucous membrane. Ulcerated in old neglected cases. Orifices of glands and lacunæ dilated. Deep layers of the urethra thickened and indurated.

Bladder.—Well-marked hypertrophy. Dilatation with sacculation occurs much later and less frequently than in prostatic obstruction, owing to youth of the patient. Inflammatory changes occur, with cystitis.

Kidneys and Ureters.—Undergo dilatation. Hydronephrosis. Pyonephrosis and pyelonephritis with advent of sepsis.

POSITION.—Just in front of the triangular ligament is much the commonest place. May be anywhere in the spongy urethra

NUMBER.—One usually, but multiple occasionally

VARIETIES.—

Annular.—Whole circumference involved, but for only a short length.

Ribbon.—As in last, but for a considerable length of the tube.

Bridled.—Only a part of the circumference affected.

Resilient.—A stricture which contracts rapidly after dilating.

Impassable.—An instrument cannot be passed

Impermeable.—Urine cannot pass.

SYMPTOMS.—

Difficult Micturition.—Stream can be started without delay after straining. Takes a long time to complete the act. Stream is poor compared with the force required

Dribbling after Micturition.—The dilated urethra between the bladder and stricture slowly empties itself and trickles on to patient's clothes.

Irritability of the Bladder.—Frequent micturition

Retention of Urine.—Acute, ending in extravasation; or chronic, with false incontinence.

Urethral Discharge of a mucopurulent, thin character

Urine at First shreddy, with small casts from the mucous glands and urethra, passed with the first portion of urine.

Urine Later, mucopurulent and ammoniacal from the changes of cystitis.

Cystitis supervenes sooner or later in untreated cases.

PHYSICAL SIGNS.—Those of retention of urine. Obstruction to passage of a urethral sound or catheter. Induration round the stricture felt externally, especially in bad strictures in the bulb.

METHOD OF PASSING INSTRUMENTS.—

Preliminary.—Patient lying on his back with head and knees raised. Cleanse the glans and meatus. Syringe the urethra with dilute antiseptic. Inject one drachm of carbolized oil.

To Ascertain the Existence of an Organic Stricture.—Pass a No. 9 Lister's bougie. If this passes easily, the stricture does not exist or is merely spasmodic.

To Find the Position of the Stricture.—Note the point at which the point of the bougie is arrested. Note the distance from the meatus.

To Find the Size of the Stricture.—Pass small filiform or olive-headed bougies. Note the size of the one which is firmly grasped by the stricture.

To Find the Length of the Stricture.—Pass acorn-headed bougie through the stricture. Withdraw it and note the distance it is withdrawn from the moment it engages the stricture until it is released.

Use soft flexible bougies à boule whenever possible. Never use any force in trying to pass a stricture.

Direction.—Pass the instrument with its handle parallel to Poupart's ligament. Then rotate the penis into midline. Raise the handle at right angles to the body, and allow the point to slip round the pubic arch.

Points where Small Instruments may be Caught.—

In one of the lacunæ, especially the lacuna magna on roof of the urethra. Keep point on the floor of the urethra as far as perineum.

At the triangular ligament. Keep the point on the roof of the urethra as it passes round the pubes.

In the sinus pocularis in floor of prostatic urethra. Keep the point on the roof of the prostatic urethra.

INJURIES AND OTHER EFFECTS WHICH MAY RESULT FROM PASSAGE OF INSTRUMENTS.—

Hæmorrhage indicates abrasion and laceration.

False Passages.—Sharp point of a metal instrument may pierce the urethral wall and re-enter the urethra or bladder, or produce a sinus. Signs: A sudden 'give', hæmorrhage, and pain. Results: Increased symptoms of retention—Peri-urethral abscess—Perineal fistula.

Cystitis or Epididymitis from sepsis.

Urinary Fever.—Sapræmia due to absorption of toxins through abrasion Septicæmia due to invasion of the kidneys by micro-organisms.

Shock may occur in nervous patients.

Suppression of Urine is rare, and supervenes on some previous disease of the kidneys.

Traumatic Stricture of the Urethra.—

CAUSE.—Follows rupture of the membranous urethra.

POSITION.—Usually at the (superficial) triangular ligament.

CHARACTER.—Dense mass of scar-like cartilage extending on both sides of the triangular ligament. Sound grates upon its surface It rapidly contracts, even if it can be dilated.

RESULTS.—Acute or chronic retention. Dilatation and ulceration of the urethra behind the stricture. Perineal abscess or fistula. Extravasation of urine.

Treatment of Organic Strictures.—

I. OF PASSABLE STRICTURES.—

1. Gradual Dilatation —Used for the majority of cases. Use Lister's bougies. Pass two or three sizes once a week. Continue until a No. 9 to 12 passes easily.
2. Continuous Dilatation.—Useful in a small stricture with severe retention. Tie in a small instrument for forty-eight hours. Then a larger size for the same time. Later practise gradual dilatation.

Treatment of Organic Strictures—Passable Strictures, ***continued.***

3. EXCISION OF THE STRICTURE.—Suitable for cases in which catheterization is badly borne. Cases which rapidly relapse after dilatation. Very dense strictures. Stricture of one inch or less in length.
   Method.—Pass a sound or Syme's staff down to or through the stricture. Expose the urethra and split it through the stricture. Cut out the stenosed part. Sew together the two healthy ends like a ribbon, allowing longitudinal incision to close spontaneously. Drain the external wound. Tie in a soft catheter.
4. INTERNAL URETHROTOMY —Inferior to excision. Used in the same class of cases Limited to those which admit a No. 4 bougie.
   Method.—Civiale's urethrotome passed through the stricture. The knife is unsheathed. The instrument is withdrawn. The knife is sheathed after cutting through the stricture. In the bulbous urethra cut towards the roof. In the penile urethra cut towards the floor. Tie in a No. 10 catheter for some days.
5. EXTERNAL URETHROTOMY.—Same class of cases as excision. The stricture must admit a No. 2 bougie.
   Method.—Pass a Syme's staff with the small end through the stricture. Cut through the stricture from behind forwards. Tie a catheter in the bladder. Leave the perineal wound open.

II. OF IMPASSABLE STRUCTURES.—

1 WITHOUT RETENTION —
   Wheelhouse's Operation.—Pass a Wheelhouse's staff. Open the urethra upon the groove. Retract the opening by the hook on the staff. Cut open the stricture from before backwards. Tie in catheter.
   Excision of the Stricture should always be attempted as a final stage where this is practicable.

2. WITH RETENTION —
   Cock's Operation.—Median perineal incision Stab incision into the the prostatic urethra, using a finger in the rectum as guide
   Urethrotomy on a More Deliberate Plan is much better.—Begin by a suprapubic cystotomy. Pass sound from bladder to stricture. Cut down upon sound in the perineum. An excision may often be done later, when the patient and the local conditions are more healthy.

Local Complications of Stricture.—

1. PERINEAL ABSCESS.—Caused by ulceration of the urethra behind the stricture. Septic urine leaks into peri-urethral structures.
   TREATMENT.—Open freely externally. Divide stricture over Wheelhouse staff. Pass gorget into bladder guided by finger in rectum. Drain bladder with a straight tube. Remove tube in 10 days. Pass instrument after 14 days.
2. PERINEAL FISTULÆ.—The result of an untreated abscess. Generally multiple, opening on to the perineum, buttocks, or scrotum. Lead into tortuous sinuses buried in dense cicatricial tissue.
   TREATMENT.—Perineal fistulæ heal spontaneously when stricture is fully dilated.
3. EXTRAVASATION OF URINE.

EXTRAVASATION OF URINE

Causes.—

RUPTURE OF THE BLADDER—extraperitoneal.

RUPTURE OF THE URETHRA BEHIND A STRICTURE.—

1. Direct Rupture following acute retention.
2. Rupture of an Ulcerated Urethra following chronic retention.

RUPTURE OF A PERI-URETHRAL ABSCESS into the subcutaneous tissues.

INJURIES TO THE URETHRA by instruments which have forced false passages round the stricture and re-entered the urethra above the stricture.

TRAUMATIC RUPTURE OF THE URETHRA.

Anatomy.—

MEMBRANOUS URETHRA is ruptured usually at or near the superficial triangular ligament

URINE ESCAPES through the triangular ligament.

Is Directed by Colles's Fascia: Forwards to the scrotum and penis; upwards to the anterior abdominal wall.

Is Bounded by Attachment of the Superficial Fascia to inner end of Poupart's ligament, and thence horizontally across the thigh to the fascia lata.

Symptoms.—Sensation of rupture when straining to relieve retention. Feeling of relief of the retention without passing urine. Rapidly fatal sapræmia or septicæmia.

Physical Signs.—Rapidly increasing boggy swelling of the perineum, external genitals, anterior abdominal wall Parts becoming dusky and then gangrenous. An emphysematous crackling is caused by the gas formed in the subcutaneous tissues. Extensive sloughing of the skin occurs if the patient recovers.

Treatment.—Early free incisions into all the affected parts. Suprapubic cystotomy with bladder drainage and irrigation. Hot hip-baths, with fomentations over all the incisions.

CHAPTER XLVII

DISEASES OF THE MALE GENITAL ORGANS

DISEASES OF THE PENIS

Malformation.—

PHIMOSIS (*see below*) and PARAPHIMOSIS (*see* p. 549).

EPISPADIAS.—Urethra opens on the dorsum of penis. Involves a torsion of the penis. Is usually associated with ectopia vesicæ.

TREATMENT by plastic operation in suitable cases.

HYPOSPADIAS.—Urethra opens on the lower aspect of the penis or in the perineum. There are three grades: (1) Meatus opens below the glans; (2) Meatus opens below the penile body, (3) Meatus opens in the perineum. The last is associated with cleft scrotum and defective development of penis and testis.

TREATMENT by plastic operations in the more severe cases.

TORSION OF THE PENIS.—The organ is twisted. Usually combined with epispadias.

DISLOCATION (very rare).—The whole organ is pulled out of its cutaneous coverings, and lies under the skin of the scrotum or groin.

DOUBLE PENIS.—Very rare.

Phimosis.—

1. CONGENITAL.—From a long, narrow prepuce. From a minute preputial orifice. From adhesion of the prepuce to the glans.

   SYMPTOMS.—Painful and difficult micturition. Ballooning of the urethra. Dribbling of urine after micturition.

   RESULTS.—Eczema of and round the genitals. Stone in the bladder. Cystitis. Dilatation of bladder, ureters, and kidneys. Hernia. Prolapsus recti.

2. ACQUIRED.—From the contraction of fissures at the orifice, from any inflammatory affection of the prepuce.

   RESULTS.—Paraphimosis. Rupture of the frænum. Liability to severe forms of venereal disease, or epithelioma.

TREATMENT.—

1. CIRCUMCISION.—In all uncomplicated cases.

   Method.—Cut off the prepuce after pulling it forward over the glans. Cut the mucous membrane of the prepuce near to the glans. Sew the edges of skin and mucous membrane with catgut.

   Errors.—Removal of too much skin: Retracts the penis—Prevents erection—Liable to ulceration. Removal of too little skin: The phimosis recurs.

2. SLITTING UP THE PREPUCE along its dorsal aspect. In cases in which a chancre or some foul secretion is concealed by the prepuce, or in which paraphimosis has occurred.

Paraphimosis.—

CAUSES.—Constriction of the glans at the corona by a contracted preputial orifice after the prepuce has been drawn back.

SIGNS AND RESULTS.—Pain and œdema. Ulceration at the constricted point. Gangrene of parts below the constriction.

TREATMENT.—Reduction under an anæsthetic. Division of the prepuce along mid-dorsal line if reduction is impossible.

Circumcision: At the time if the parts are healthy. Later if much inflammation exists.

Priapism.—Continuous erection in the absence of sexual desire.

CAUSES.—Venereal excess. Injury or thrombosis in corpora cavernosa. Phimosis or stone in the bladder (children). Leukæmia. Injuries to the cervical spinal cord

SYMPTOMS.—Pain. Mental distress In nerve cases there is turgescence without any pain or rigidity.

TREATMENT.—In nerve cases leave it alone.

SEDATIVES: Bromides, antimony, morphia, etc.

INCISIONS into the corpora cavernosa

Tumours.—

SEBACEOUS CYSTS

HORNS.—Growing from a ruptured sebaceous cyst.

PAPILLOMA.—Common in gonorrhœa. Usually grow inside the prepuce, or from the glans. Are soft, friable, dendritic masses. There is no infiltration of their base.

Treated by removal with the scissors, touching the base with the cautery. Or by appplication of calomel

EPITHELIOMA (*see below*)

SARCOMA (very rare)

Epithelioma.—

AETIOLOGY.—Men over 40 Almost always occurs in conditions of phimosis Unknown among Jews. Any ulcer, wart, or fissure may give rise to it.

POSITION.—Generally starts in the sulcus behind the corona

CHARACTER —(1) Cauliflower-like mass of bleeding warts with indurated base; or (2) A deep ulcer with indurated base and edges, often concealed by the phimosis, with foul sanious discharge.

PROGRESS.—May form an advancing fungating mass or a deep, foul ulcer. Either kind may slough through the prepuce. The urethra is often opened behind the meatus, causing a urinary fistula. Inguinal glands enlarge and often suppurate. The lymphatics may stand out as red lines. A solid œdema may result from lymph obstruction.

DIAGNOSIS.—

PAPILLOMATA.—Have no induration.

CHANCRE, or any indurated ulcer with phimosis.—If it does not quickly yield to treatment, slit up the prepuce, remove, and examine.

TERTIARY ULCERATION (so-called relapsing chancre).—Follows a primary chancre. Yields to the action of iodides., Is avascular There is little or no new growth at the edges.

Epithelioma of the Penis, ***continued.***

TREATMENT.—

AMPUTATION THROUGH THE BODY OF THE PENIS.—In early cases confined to the glans.

AMPUTATION OF THE WHOLE PENIS.—In advanced cases. The scrotum is split. The crura are dissected away from the pubes.

COMPLETE REMOVAL OF ALL GLANDS from both groins.

EPITHELIOMA OF THE SCROTUM

(*'Chimney-sweep's Cancer'.*)

General Characters.—This is a carcinoma of the skin of the scrotum, and it presents as a typical malignant ulcer. The progress is slow, and the inguinal glands are affected late.

Treatment.—Complete removal, together with the inguinal glands.

CONGENITAL ABNORMALITIES OF THE TESTIS

Polyorchism (more than two testes), **Anorchism** (no testis), and **Monorchism** (one testis), are all very rare.

Retained Testis.—The testis remaining in the abdomen or inguinal canal is common.

DEGREES OF RETENTION.—

Abdominal.—It is attached to the posterior abdominal wall—i.e., true undescended testis.

Iliac.—It lies deep to the internal abdominal ring.

Inguinal.—It lies in the inguinal canal

Retractile.—Lies in the inguinal canal, but can be pressed down into scrotum.

Other Positions.—May be superficial to inguinal canal, pubic, or femoral.

STRUCTURE AND SIGNS.—A soft or tender lump exists at the internal ring or in the canal. The scrotum is ill-developed, and empty on that side. The testis is usually soft, ill-developed, and functionless.

COMPLICATIONS.—Hernia usually coexists. Complete atrophy of the testis. Torsion and gangrene Orchitis. Hydrocele or hæmatocele. Malignant disease.

DIAGNOSIS from: Hernia, hydrocele of the cord, and other inguinal swellings

Absence of the testis from the scrotum is the main clue.

TREATMENT.—

By Hormones.—A gonadotropic hormone extracted from the urine of pregnant women, known as pregnyl, is given by intramuscular injection. 500 units twice a week for 3 to 6 months. It cures all merely retractile testes, but these would descend naturally. It should be used at the ages of 10 to 14. Is more likely to succeed in bilateralcases.

By Operation.—Torek's operation: Testis is brought down to scrotum, and scrotal wall is incised. Deep fascia on inner side of thigh is now exposed, and raw edge of scrotum and testis is sewn to this (*Fig.* 183). Three months later testis and scrotum are freed from the thigh.

Ectopia Testis.—Testis leaves the inguinal canal in an abnormal position. Various positions: (1) Perineal; (2) Crural; (3) Near the anterior superior spine; (4) Pubic; (5) Prepenile.

TREATMENT.—Removal if it is painful or inflamed.

Torsion of the Spermatic Cord.—The testis is rotated once or twice and the cord is twisted. The twisted cord causes strangulation of the vessels. It probably arises from conditions of imperfect descent and development.

Anatomy.—Great swelling and ecchymosis of the epididymis. The body of the testis is comparatively normal. Possible gangrene of the whole organ.

Symptoms.—Sudden sickening pain in the groin. Great swelling of testis and cord below strangulation. Vomiting and collapse.

Diagnosis.—From strangulated hernia. By the following points: Any evidence of non-descent of the testis. The epididymis being in front of the testis. A normal part of the cord above the swelling. Absence of constipation.

Treatment —Removal of the testis and cord Replacement is seldom desirable or justifiable.

VARICOCELE

Definition.—A marked hypertrophy and varicosity of the spermatic veins.

Aetiology.—It always occurs on the left side. The reasons for this are unknown, but may be: (1) Lower position of testis; (2) The opening of the left spermatic vein into the renal vein instead of direct into the vena cava; (3) A congenital anomaly.

Symptoms.—Often there are none. A dragging pain, especially after excretion or copulation When once the patient's attention has been attracted to it, hypochondriacal symptoms are common. It debars from public service.

Signs.—The large mass of veins is easily felt in the scrotum. They feel like a 'bag of worms' They empty when the patient lies down, and they give an impulse on coughing or straining. The testis is often small and soft.

Treatment.—Suspensory bandage is all that is required for the vast proportion of cases. Excision of the veins in front of the vas can be performed for the worst cases.

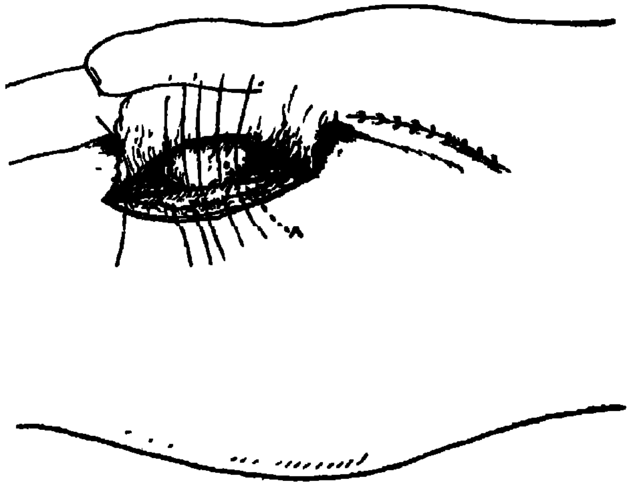


Fig. 183.—Torek's operation for undescended testicle. A, Testicle sutured to fascia of thigh.

HYDROCELE

Definition.—A collection of serous or spermatic fluid connected with the testis, cord, or their coverings.

Varieties (*Fig.* 184).—

I. VAGINAL, i.e., in a normal or abnormal tunica vaginalis.—
 1. CONGENITAL.—In the whole funicular and vaginal tunics, whose cavity communicates with the peritoneum.
 2. INFANTILE.—Similar to the congenital, but shut off from the peritoneum at the internal ring.
 3. BILOCULAR.—In an abnormal, bilocular, funicular sac.
 4. ACQUIRED:—
  a. Primary.—A chronic affection of the tunica vaginalis itself.
  b. Secondary.—May be acute or chronic, secondary to disease or injury of the testis or epididymis, or following operations such as for varicocele or inguinal hernia.

II. HYDROCELE OF THE CORD, usually funicular.—
 1 ENCYSTED HYDROCELE OF THE CORD.—Arises in an unobliterated part of the funicular process shut off from the tunica vaginalis below.
 2. FUNICULAR HYDROCELE.—Arises in the patent funicular process.
  a. Congenital.—Communicates with the peritoneum.
  b. Infantile.—Is shut off from the peritoneal cavity.
 3. DIFFUSE HYDROCELE OF THE CORD —A condition of lymphatic œdema.

III. HYDROCELE OF THE TESTIS AND EPIDIDYMIS.—
 1. ENCYSTED HYDROCELE OF THE EPIDIDYMIS —From a dilatation in the spermatic ducts.
 2. ENCYSTED HYDROCELE OF THE TESTIS —Arises beneath the tunica albuginea.

IV. HYDROCELE OF A HERNIAL SAC —In infancy this will be a congenital hydrocele. In adults a similar condition may arise in the sac of a hernia after its contents have been reduced

Primary Chronic Vaginal Hydrocele.—

CAUSES.—Practically unknown Probably some inflammatory thickening of the tunica vaginalis.

STRUCTURE.—Tunica vaginalis becomes thickened in proportion to the age of the hydrocele. It may become fibrous, cartilaginous, or calcified. Fibrous or warty growths may be found free in the cavity or growing from the surface of the tunic.

The fluid is usually about 4 to 12 oz.; sp. gr. 1025, neutral reaction, 6 per cent albumin. Coagulates when mixed with a little blood. It is usually clear straw-coloured. It may be green (blood pigments), turbid (mucus), or shimmering (cholesterin crystals).

ANATOMY.—The enlarged tunic surrounds the testis, which lies at the back and lower part of the swelling (*Fig.* 185, *a*) The enlargement distends the scrotum, and has but little or no tendency to travel up the cord.

SIGNS AND SYMPTOMS.—A scrotal swelling with the following characters: Large size, but of slow formation. Absence of much pain or tenderness. Feels smooth on the surface, is globular or pyriform. Elastic or fluctuating in proportion to thinness and tenseness of the sac.

Translucency. This may be absent: when the fluid contains much blood or mucus; if the tunica vaginalis is very thick; if many adhesions exist inside the sac.

It is dull to percussion. There is no impulse on coughing. It is bilateral in about one case in five.

DIAGNOSIS.—

HERNIA.—In this there are: A swelling which extends into the inguinal region. Impulse, reducibility, gurgling, resonance. An irreducible epiplocele is the most difficult to distinguish. The obscurity of the cord, the doughy feel, and the opacity, are the chief points. The two conditions may coexist.

HÆMATOCELE.—Feels harder, heavier, and less elastic. Is not translucent. Often a history of injury. There may be some ecchymosis.

SOLID ENLARGEMENTS OF THE TESTIS AND EPIDIDYMIS.—These are opaque and do not fluctuate. The outline of the body and epididymis is usually to be felt. Pain and tenderness are generally marked. The cord is often thickened. In inflammatory enlargements of the organ a secondary hydrocele may be present, but inflammatory signs are evident

A sarcoma is the most difficult to distinguish, because it is often so elastic.

OTHER VARIETIES OF HYDROCELE —Hydroceles of the cord and epididymis and testis leave the body of the testis unobscured. Congenital and infantile hydrocele run up to the internal ring, the congenital variety being reducible.

TREATMENT.—

TAPPING.—Make sure of the position of the testis. Drive the trocar upwards and backwards so as to clear the testis. It has to be repeated every few months.

INJECTIONS of strong antiseptics after tapping. Linimentum iodi ℥ij, water ℥iv; or pure carbolic and glycerin, āā ℥j.

EXCISION of part or whole of the sac Especially indicated when the fluid reaccumulates very quickly after tapping, or when the sac is thick

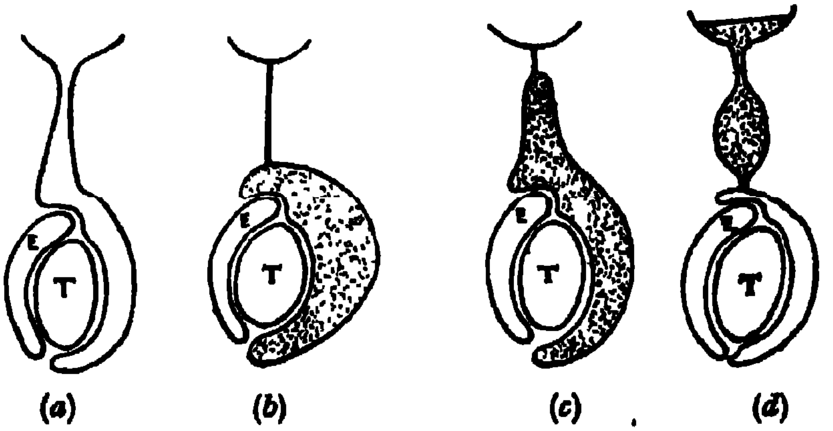


Fig. 184.—Diagrams of hydrocele: (*a*) Congenital hydrocele; (*b*) [illegible]inal hydrocele; (*c*) Infantile hydrocele; (*d*) Congenital funicular hydrocele. T [illegible]ody of testis; E, Epididymis.

Primary Chronic Vaginal Hydrocele—Treatment, ***continued.***

EVERSION OF THE SAC.—The hydrocele is exposed and an incision made into it just large enough to allow the testicle to be brought out through it. The sac is now turned inside out and secured with a stitch. The everted sac and testicle are then returned to the scrotum.

Secondary Chronic Hydrocele.—Of small size, merely obscuring the testicular outline. Secondary to syphilis, tubercle, or chronic inflammation.

TREATMENT.—As for the primary disease. Firm strapping may be useful.

Acute Hydrocele.—Always secondary to some injury or disease of the testis. Gonorrhœa, traumatic orchitis, or wounds may cause it. Usually is absorbed spontaneously, leaving adhesions. Very rarely suppurates.

TREATMENT.—As for the primary disease.

Congenital and Infantile Hydrocele.—Translucency is well marked. The swelling extends into the groin Congenital hydrocele is slowly reducible.

TREATMENT.—Usually disappears spontaneously Tapping (in infantile), followed by a truss. Injections are dangerous Operate only if a hernia coexists.

Bilocular Hydrocele.—A form of infantile hydrocele. The open funicular process has a secondary pouch opening from it. This may lie between the peritoneum and muscles, between the muscles, or between the skin andmuscles

TREATMENT —By open incision, dissecting out as much as possible

Hydrocele of the Cord.—A fluid swelling above and separated from the testis. Congenital form is reducible into the peritoneum Infantile form extends into the inguinal canal, but is irreducible Encysted form is a globular enlargement at one part of the cord It may be multiple. Commoner on right side than on left.

In addition to translucency and fluctuation, all these are distinguished by moving with traction on the testis And usually an upper limit can be defined, so distinguishing them from herniæ.

TREATMENT.—Acupuncture or excision.

Encysted Hydrocele of the Epididymis.—

FORMED BY A DILATATION OF ONE OF THE DUCTS, or fœtal tubes, at the head of the epididymis.

USUALLY CONTAINS ALTERED SPERMATIC FLUID. It is alkaline with little or no albumin. Opaque, milky appearance It effervesces with acetic acid. It contains many living or degenerate spermatozoa

It forms a swelling ABOVE AND DISTINCT FROM THE TESTIS and tunica vaginalis.

Rarely contains more than a few ounces of fluid

Encysted Hydrocele of the Testis.—Situated beneath the tunica albuginea. Usually of quite small size. Resembles the last variety when it becomes large

TREATMENT —Both of the last two conditions are treated by acupuncture or excision.

HÆMATOCELE

Definition.—A blood collection in the tunica vaginalis or cord.

Varieties.—Vaginal: the only common variety. Diffuse in the cord: from ruptured vessel Encysted in testis, epididymis, or cord: arises from bleeding into the corresponding hydrocele.

Causes.—Contusion of the testis. Contusion or puncture of a hydrocele. Secondary to malignant disease.

Structure.—Massive thickening of the walls of the tunica vaginalis with patches of cartilage or calcareous material. Filled with old and laminated clot, with, perhaps, a little fluid. The testis is often degenerated.

Signs and Symptoms.—An oval tumour, which envelops both testis and epididymis. It is not translucent, and feels solid or doughy. There is history of either a trauma or a hydrocele. Ecchymosis may be well marked in recent cases. The cord is not thickened. The size is stationary or nearly so.

Diagnosis.—

From Hydrocele.—This is fluctuating and translucent. Puncture yields clear fluid.

From Traumatic Orchitis.—Diagnosis is very difficult. In this the outline of testis and epididymis can often be made out. Tenderness is much more marked.

From Sarcoma, Gumma, or other New Growth.—These steadily increase in size. In their early stages the testis and epididymis can be distinguished.

In all doubtful cases it is better to explore.

Treatment.—Palliative in the acute cases after trauma—rest, elevation, and lotions. Operative in chronic cases. Turn out the clot, and excise the sac. Castration in old men with degenerate testis and much pain.

INFLAMMATORY DISEASES OF THE TESTIS AND EPIDIDYMIS

Varieties.—

EPIDIDYMITIS.—

Acute or Chronic.—(1) Simple. (2) Pyogenic (3) Gonorrhœal. (4) Tuberculous (5) Syphilitic.

ORCHITIS.—

Acute —(1) Traumatic. (2) Complicating a specific fever.

Chronic —(1) Resulting from the acute form. (2) Syphilitic (3) Tuberculous.

EPIDIDYMO-ORCHITIS.—Begins as an orchitis. Usually traumatic.

EPIDIDYMITIS

Acute Epididymitis.—

CAUSES —Some primary inflammation of the posterior urethra, prostate, or bladder, viz.: Gonorrhœa—Gouty urethritis—Septic catheterization —Stricture—Prostatitis—Cystitis—*B. coli* pyelitis. It also occurs fairly commonly after enucleation of the prostate.

ANATOMY.—Epididymis is much swollen, the tail being chiefly affected (*Fig.* 185, *b*). Vas is generally swollen. Tunica vaginalis may be inflamed, and its fluid increased to form an acute hydrocele.

SIGNS AND SYMPTOMS.—A boat-shaped swelling behind the testis which is very tender, and most marked at the lower end. The scrotum is red, inflamed, and often adherent. There may be a fluid swelling obscuring the testis, from affection of the tunica vaginalis. The vas is swollen, tender, and painful. General malaise, with rise of temperature.

Acute Epididymitis—Signs and Symptoms, *continued*.

Extreme tenderness. Walking with the legs abducted. Acute stage lasts one or two weeks. Often leaves a nodular thickening in the globus minor. The urethral discharge generally diminishes in acute stage. One side is affected after the other.

SEQUELÆ.—Chronic epididymitis. Abscess (very rare). Sterility in bilateral cases.

TREATMENT.—Rest in bed, with support to the scrotum. Evaporating lotions. Hot fomentations if great pain exists. Do not give any urethral injections.

Chronic Epididymitis.—

VARIETIES.—Simple: is commonly gonorrhœal. Tuberculous. Syphilitic (*see below*).

CAUSES.—The same as in acute epididymitis. Generally is the remains of an acute attack. Sometimes is chronic from the first.

SYMPTOM —Dragging pain in the testis.

SIGNS.—An induration and swelling of the epididymis and vas. Most marked at the tail, i.e., the globus minor. May affect the entire epididymis. The globus minor is affected first in gonococcal infections, the globus major in syphilitic infections.

RESULT.—Often produces sterility if bilateral

TREATMENT.—Strapping and suspension of the testis.

ORCHITIS

Whereas epididymitis seldom spreads to the body of the testis, inflammation of the body often spreads to the epididymis.

Traumatic Orchitis.—Often affects both epididymis and testis. Results from crushes and contusions. Consists of hæmorrhagic and fibrinous masses in the substance of the testis and epididymis The organ is enlarged, very hard, nodular, and tender. Usually leaves a chronic nodular thickening. May result in atrophy.

TREATMENT.—By strapping and suspension

Acute Orchitis.—

CAUSES.—Traumatism. Infective fevers· Mumps—Enteric—Variola—Tonsillitis—Scarlet fever—Malaria—Influenza. Rheumatism—Gout.

SIGNS AND SYMPTOMS (of the only common form, viz, that in mumps). —Occurs in young adults. Begins about a week after the parotitis. Testis swells to two or three times its natural size (*Fig.* 185, *c*). It retains its shape, and is firm and elastic. It is intensely tender and painful. One side may be affected after the other. High fever and prostration occur for a few days, but soon subside. Suppuration almost never occurs in parotitic forms, but may in variola, enteric, rheumatism, or gout. Atrophy is very rare as a sequel.

TREATMENT.—Constitutional treatment, rest in bed, etc Cooling lotions in most cases. Fomentations in gout or rheumatism

SYPHILIS OF THE TESTIS

Varieties.—(1) Congenital. (2) Acquired: (*a*) Secondary, and (*b*) Tertiary.

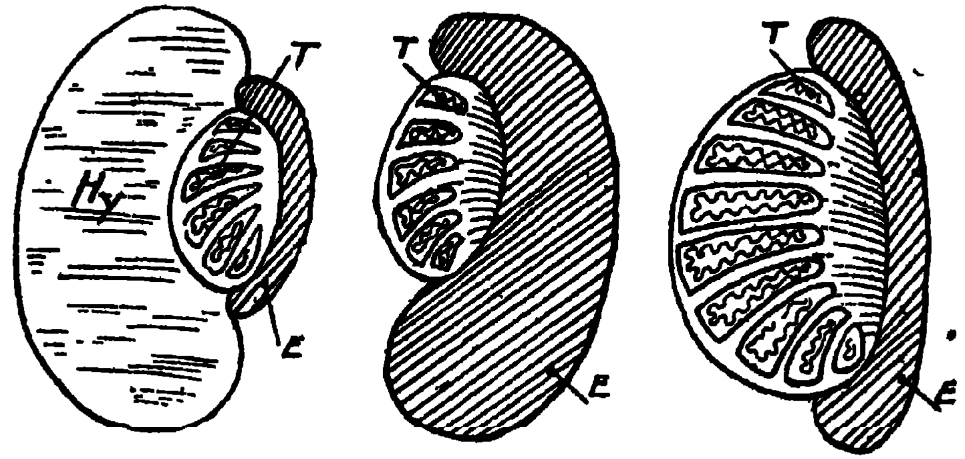
1. Congenital.—Occurs as a bilateral gummatous orchitis. Syphilis is practically the only cause of testicular enlargement before the age of six months. Later the testicle shrinks and becomes fibrotic, so that in an adult a shrunken testicle without history of previous injury is very suggestive of congenital syphilis. Such persons are impotent (Hutchinson's tetrad—'the halt; the blind; the deaf; the impotent'). May occur as a gummatous infiltration about puberty

2. Acquired.—

a. SECONDARY.—Transitory symmetrical epididymitis mainly affecting the globus major—'reminders'; Readily disappears after giving mercury, but may leave a fibrous nodule.

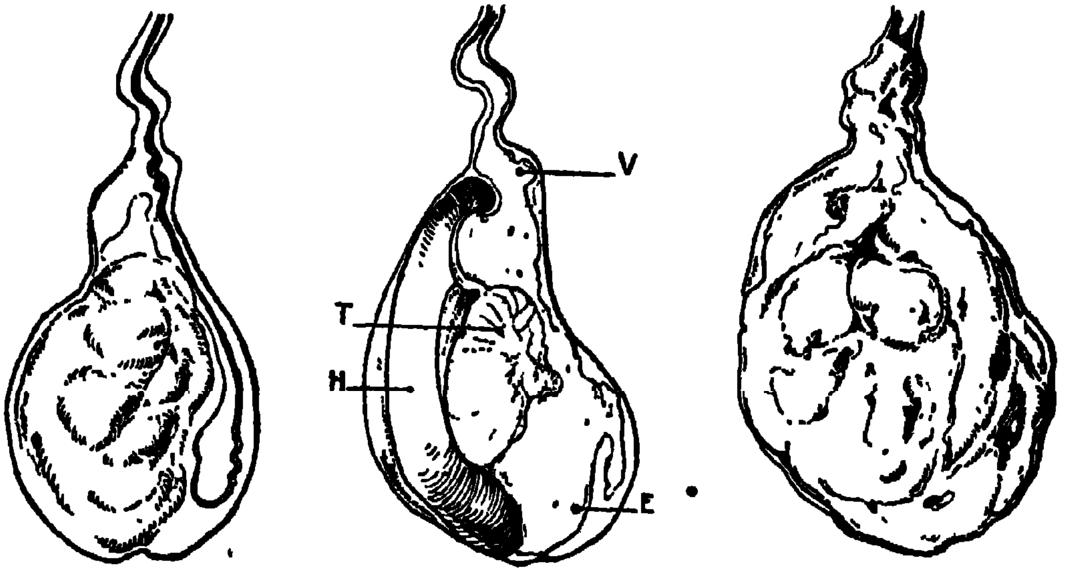
b. TERTIARY.—

PATHOLOGY.—May occur as a localized gumma with interstitial orchitis, or as a gummatous infiltration (*Fig.* 186, *a*). The testicle becomes



(*a*) (*b*) (*c*)

Fig. 185.—Affections of the testicle. (*a*) Hydrocele of the tunica vaginalis. (*b*) Epididymitis. (*c*) Orchitis. T, Testis, E, Epididymis, Hy, Hydrocele.



(*a*) (*b*) (*c*)

Fig. 186.—Affections of the testicle. (*a*) Gumma, affecting body, but not vas or epididymis. (*b*) Tubercle: H, Hydrocele; T, Body of testis, unaffected; E, Epididymis and V, Vas, affected. (*c*) New growth, affecting body, epididymis, and vas.

Tertiary Syphilis of the Testis—Pathology, ***continued.***

replaced by fibrous tissue in which gummatous caseation occurs. This adheres to the scrotum and breaks through anteriorly, forming an ulcer or fungus testis. There may be a secondary hydrocele.

SIGNS.—Slow, steady enlargement of body of the testis. Mass is nodular, heavy, painless, and without testicular sensation. A secondary hydrocele may coexist. An ulcer or fungus may be present. There is a slight diffuse thickening of the cord (cremaster and vessels).

TREATMENT.—Iodides, with mercury. If evidence of caseation exists, remove the testicle so as to avoid fungation.

TUBERCULOUS DISEASE OF THE TESTIS

Aetiology.—Occurs in young adults, or even in young children. Is secondary to disease elsewhere, especially bladder, prostate, vesiculæ, kidneys, or lungs.

Pathology.—

EPIDIDYMIS is affected first almost invariably.

THE GLOBUS MAJOR is the earliest seat of the disease. If the globus minor is first affected, then the disease is probably secondary to tuberculous disease of the seminal vesicles or prostate.

NODULAR INFILTRATION, CASEATION, and FISTULA FORMATION succeed each other. In chronic or cured cases, and especially in children, a hard fibrous mass of scar tissue is left permanently.

THE DISEASE MAY EXTEND into the following tissues: Vas deferens, vesiculæ seminales, prostate, tunica vaginalis (producing hydrocele, or obliteration by adhesions), scrotum (forming a fistula or fungus), the body of the testis (rarely), the bladder, and the kidneys. (*Fig.* 186, *b*.)

Signs.—EPIDIDYMIS SHOWS NODULAR THICKENING, first about the head, then the tail, then the body. These are hard and not inflamed at first. After several weeks, or months, softened areas are felt.

THE SKIN OF THE SCROTUM becomes red and adherent, and the abscess breaks, discharging tuberculous pus.

A FISTULA or FUNGUS TESTIS results.

The tunica vaginalis at first is filled by a small hydrocele Later it is usually obliterated by adhesions.

The fungus, or fistula, may open behind without involving the tunic, or it may first invade the tunic, and then open in front.

The opposite testis generally is involved later on, and will present an earlier stage of the same disease.

THE VAS is felt as a thickened cord.

THE PROSTATE AND SEMINAL VESICLES may be felt to be thickened and nodular per rectum.

Course.—As in tuberculous disease elsewhere, the rate of progress varies much. Usually it is a slow process, lasting many months. Death occurs generally by infection of the bladder, kidneys, or lungs Cases may become chronic, and a curative fibrosis take place. In this case the ducts will almost certainly be obliterated, and therefore for reproductive purposes the testis is functionless.

Treatment.—

MEDICAL TREATMENT in chronic or slight cases.

Prophylactic ligature of the vas deferens on the opposite side should be performed to prevent bilateral disease.

Epididymectomy.—In bilateral cases, in order to preserve the general influence of the glands.

As extensive removal of the vas as possible should always be done, and in some cases, the iliac and lumbar lymph-glands.

Diseased vesiculæ seminales may be removed by the perineal route if the bladder and kidneys are sound.

If the bladder and kidneys are affected it is useless to operate, except as a palliative measure for fungus or fistulæ.

NEW GROWTHS OF THE TESTIS

Pathology.—All new growths of the testicle must be regarded as malignant. Tumours described as fibroma, adenoma, chondroma, lipoma, and myoma are either the predominating constituents of a mixed growth, or are so rare as to be of no clinical value. A teratoma may remain stationary for years, but eventually takes on rapid growth.

VARIETIES.—

1. Typical Teratoma or Fibrocystic Disease —This tumour contains all three primary layers—epiblast, mesoblast, and hypoblast.
2. Atypical Teratoma —This is the malignant stage of the typical teratoma. One of the primary layers develops and predominates at the expense of the other two layers.
3. Carcinoma, Seminoma, or Spermatocytoma —This is a spheroidal-celled tumour arising from the spermatocytes of the second order in the germinal layer of the seminiferous tubules.
4. Chorion Carcinoma.—This probably arises by a process of metaplasia from the epiblast of a teratoma.
5. Sarcoma.—A pure sarcoma is very rare. The majority of the cases described are probably atypical teratomata in which the mesoblastic tissue predominates

Aetiology.—The average age is about 30.

Symptoms.—In the early stages there is neither pain nor discomfort, but a steady increase in size of the testicle. The enlargement does not at first involve the epididymis, which can be felt separate from the testicle. Rounded nodular swellings may be felt as the tumour increases in size (*Fig.* 186, *c*) The testicle feels heavy and is usually very hard and insensitive If rapidly growing with extensive degeneration it may give an impression of fluctuation. Metastases occur in the lumbar aortic glands and form a deep-seated mass at or above the level of the umbilicus. Metastases may occur in the lungs.

Diagnosis.—

CHRONIC HÆMATOCELE.—May have a history of injury and subsequent increase in size, with acute pain.

GUMMA.—Rapid improvement with antisyphilitic treatment.

HYDROCELE —If old may not be translucent. History of tapping may confirm diagnosis.

Often the diagnosis is impossible without exploration.

Treatment.—Castration is of little use and is now abandoned. Prophylactic deep X-ray therapy is now given in early cases. Prognosis of most cases is bad, but castration may be performed, together with retroperitoneal removal of the lymphatics and glands up to the level of the renal vein, if fungation of the growth is about to occur.

CHAPTER XLVIII

AMPUTATIONS

Introductory.—Removal of a limb or of a part of it was a very common operation in the old days before the advent of aseptic surgery made conservative measures generally possible. The classical amputations of a generation ago were invented when speed was one of the chief considerations, the patient not having the advantage of anæsthesia. In modern times amputations are comparatively uncommon, and the great majority of the older methods have been abandoned in favour of a few simple types. This is because the art of the limb-maker has improved, and it has been found, especially since the experiences of the last war, that only those types of amputation after which an artificial limb can be fitted are worth while doing. These have again been modified by experiences of the present war.

Indications for Amputation.—

TRAUMA.—Crushes of a part of the limb. Extensive tearing of the soft parts, as in machinery accidents. Open fractures, with concomitant injury to joints or blood-vessels. War wounds.

GROSS DEFORMITY.—Mal-united fractures, especially when involving joints. Rarely in infantile paralysis or Charcot's disease.

GANGRENE.—All types of gangrene require removal of the dead part of the limb. But in some cases of senile gangrene, if painless, it is better to allow the part to be separated by natural ulceration, because surgical amputation would require removal high up through healthy tissues.

SEPSIS OR TUBERCULOSIS.—When the bones and joints are infected and fail to recover with conservative treatment.

CHRONIC ULCERATION (of the leg).—When an ulcer has destroyed more than half the circumference of the leg, or short of this has relapsed after conservative or plastic treatment

MALIGNANT DISEASE.—Usually sarcoma of bone.

PRIMARY OSTEOGENIC SARCOMA.—High amputation of the whole limb.

OSTEOCLASTOMA.—(If conservative measures have failed.) Local amputation.

CHONDROMA.—When the tumour has destroyed the usefulness of the limb.

EPITHELIOMA OF THE SKIN.

Site of Amputation.—

Above the disease to be removed.

Through healthy tissues with good circulation.

In upper limb, so as to save any part of the digits or palm of the hand which is viable.

In lower limb, so as to adapt the stump to the best form of artificial leg.

Technique of Amputation.—

INCISION.—

- CIRCULAR OR ELLIPTICAL, TURNING UP A CUFF OF SKIN.—When bone is central and when there is to be no weight-bearing.
- RACKET-SHAPED.—At the shoulder or hip when main blood-vessels have to be secured as the first stage of the amputation.
- WITH FLAPS.—Where the bone is not centrally placed and where it is desirable to leave the scar at the side and not at the end of the stump.

LENGTH OF SKIN CUFF OR FLAPS.—

- There must be enough skin to cover the stump without tension.
- Flaps must together exceed in length the diameter of the limb at the point of bone section.
- Skin cuff must exceed in length the half diameter of the limb at point of bone section.

HÆMOSTASIS.—

- PRELIMINARY TOURNIQUET in all cases except the shoulder and hip.
- TIE ALL OPEN VESSELS after amputation, then remove tourniquet and tie any bleeding points.

MUSCLES.—Cut all muscles with oblique clean cuts, but do not sew together the cut surfaces of opposite muscles over the ends of the bone.

NERVES.—Cut the nerve level with the muscles, do not crush or tie.

BONE.—Divide the periosteum lower than the bone, turn back a cuff, and suture over the bone, so as to avoid the growth of periosteal bone-spurs.

CLOSURE OF WOUND.—

- SEW UP, SEPARATELY, periosteum, muscles, fascia, and skin, so as to obliterate dead spaces and to provide a snug covering to the bone.
- DRAINAGE is indicated when oozing cannot be checked or when sepsis has been present

Guillotine Amputation.—This was used extensively in the 1914–1918 war for fulminating septic conditions. It is probably never justified. The limb was 'chopped off' by a circular cut which divided all tissues at the same level The dressing of the large wound is very painful, and a secondary amputation is always necessary, and it is very easy to cut transfixion flaps at the initial operation and so save all this subsequent treatment. If necessary, the flaps can be sewn back for a few days.

The object of this amputation can be attained by the ordinary circular amputation, dividing the bone higher than the skin by rather more than half the limb diameter.

The skin is pulled down by adhesive plaster and the wound packed with flavine gauze or vaseline gauze.

After-treatment.—

DURING HEALING OF THE WOUND.—The limb should be kept in a suitable splint, e.g., a Thomas's, so as to prevent contracture (this applies especially to amputations below the knee).

WHEN THE WOUND HAS HEALED.—In the case of the lower limb the patient should be encouraged to walk, first with a temporary artificial limb. Stump should be bandaged to make it conical and exercises be performed.

Amputations—After-treatment, *continued.*

THE ARTIFICIAL LIMB should be fitted about 1 to 3 months after amputation, when the stump has shrunk to its final size and is free from tenderness.

WHEN WOUND BECOMES SEPTIC.—When amputation is done for septic conditions, e.g., open fractures or osteomyelitis, the wound is liable to become septic. It is important to treat such a condition promptly and efficiently, in order to avoid a re-amputation at a higher level.

REMOVE THE STITCHES FREELY until the depths of the wound can be packed with gauze soaked in flavine solution (1–1000) or permeated with Carrel-Dakin tubes.

APPLY ADHESIVE PLASTER TO THE SKIN and fix the ends of the strips by weight traction (2 to 4 lb.) or tie them to the end of a Thomas splint placed over the limb. In this way retraction of the soft parts will be prevented and the amount of bone lost by necrosis will be reduced to a minimum. (*Fig.* 187.)

SPECIAL AMPUTATIONS

Fingers and Thumb.—

FOR PRESERVATION OF A WORKING DIGIT.—If the digit has to be preserved—e.g., in the thumb, or index finger, or when other fingers have been lost—the amputation is done by *a long palmar flap* which covers the end of the stump (*Fig.* 188, A)

FOR REMOVAL OF A DIGIT.—When the digit has to be taken away because of disease or because it is useless, then *a racket-shaped incision*, with the handle of the racket on the dorsum, is the best (*Fig.* 188, B). The digit is disarticulated at the knuckle-joint. Rarely the corresponding metacarpal bone is also removed (*Fig.* 188, C).

Hand.—As amputation of the hand is always done for injury or gangrene, no formal lines can be laid down Cut the flaps where healthy skin exists, choosing that from the palmar surface by preference (*Fig* 188, D)

Forearm and Arm.—*A circular or elliptical incision* is the rule. A cuff of skin is turned up and the muscles divided at a level higher than the skin (*Fig.* 189, C).

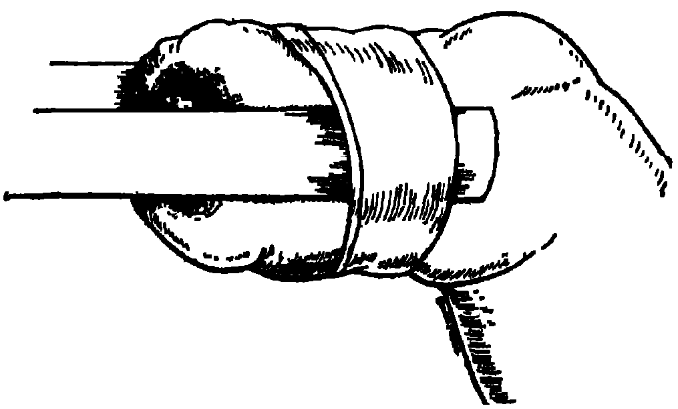


Fig. 187.—Adhesive plaster applied to leg for traction on the soft tissues in septic cases.

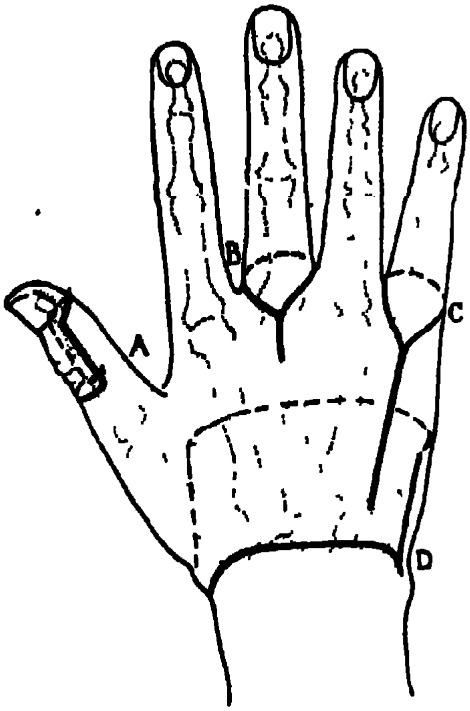


Fig 188—Amputations of the fingers and hand, A, Amputation of part of the thumb by a long palmar flap, B, Racket-shaped incision for amputation of a digit, C, The same, including the metacarpal bone, D, Amputation of the hand by a long palmar and short dorsal flap.

Fig 189—Amputations of shoulder arm, and forearm A, Amputation at the shoulder by a racket-shaped incision, B, Amputation through the middle of the arm by a circular incision, C, Amputation through forearm by a circular incision.

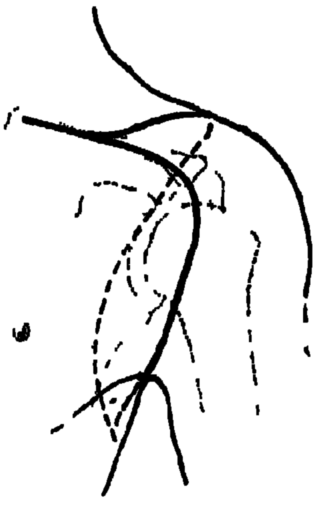


Fig. 190.—Forequarter amputation by a racket-shaped incision.

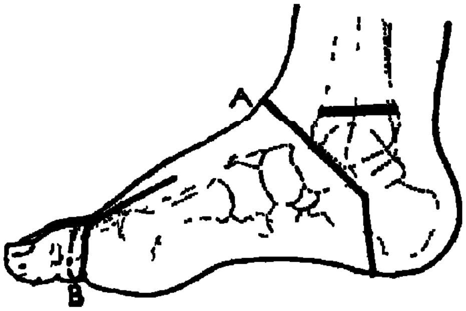


Fig 191—Amputation of the toes and foot A, Incision for Syme's amputation of the foot, B, Racket-shaped incision for amputation of a toe.

Special Amputations—Forearm and Arm, *continued.*

If removal of the whole forearm is required, it is better to amputate above the condyles rather than through the elbow, because the lower end of the humerus forms a bulbous stump, and because the artificial limb can have its joint at the level of the normal elbow (*Fig.* 189, B).

Shoulder.—Use *a racket-shaped incision.* Begin at the coracoid process and cut down along the anterior border of the deltoid. Find and ligature the great vessels. Cut round the arm at the level of the anterior axillary fold (*Fig.* 189, A).

Forequarter Amputation.—Removal of the outer half of the clavicle and the scapula with the arm.

Make *a racket-shaped incision* with the handle along the line of the clavicle, the circular part surrounding the shoulder at the level of the axilla (*Fig.* 190). Divide the clavicle at its inner third and retract the outer portion. Expose and ligature the subclavian artery and vein. Inject the brachial plexus with 2 per cent novocain. Turn back the skin flaps. Cut through the muscles attaching the arm to the chest.

Toes and Front of Foot.—

REMOVAL OF SEPARATE DIGITS is done by *a racket incision,* through the metatarso-phalangeal joint (*Fig.* 191, B).

REMOVAL OF ALL THE TOES is sometimes called for in cases of frostbite.

LISFRANC'S AMPUTATION consists of making *a long plantar flap* with removal of all the metatarsals.

MIDTARSAL AND SUBASTRAGALOID AMPUTATIONS are seldom done, because of the loss of control of the bones forming the stump, which tend to become dislocated backwards and plantar flexed.

Whole Foot.—Syme's amputation. This amputation is still being used successfully. Difficulty is fitting with subsequent artificial boot, which is rather heavy and cumbersome.

Two points are taken: (1) The tip of the external malleolus; (2) A point one finger-breadth below and behind the tip of the internal malleolus. These are joined by two cuts down to the bone, one across the front of the ankle and the other below the heel. The foot is disarticulated in front at the ankle. The calcaneum is dissected out from the heel flap, dividing the tendo Achillis. The soft parts are turned up from the malleoli and the ends of the tibia and fibula cut off (*Fig.* 191, A).

Leg.—

AT SEAT OF ELECTION (i.e., 5 to 7 in. below the knee.)—This is the operation of choice for all conditions requiring removal of the lower part of the leg. Many consider that it should supersede Syme's amputation because it is better suited for an artificial limb

Formation of Flaps.—If there is ample healthy skin, the long external flap is best. Otherwise equal lateral flaps will serve. The combined flaps should exceed in length the diameter of the calf at the level of the bone section. *The external flap* should contain all the muscle down to the interosseous membrane, and round the fibula, and should include the anterior tibial artery. *The incision* for the external flap begins at the crest of the tibia about 6 in. distal to the tubercle, and ends in the

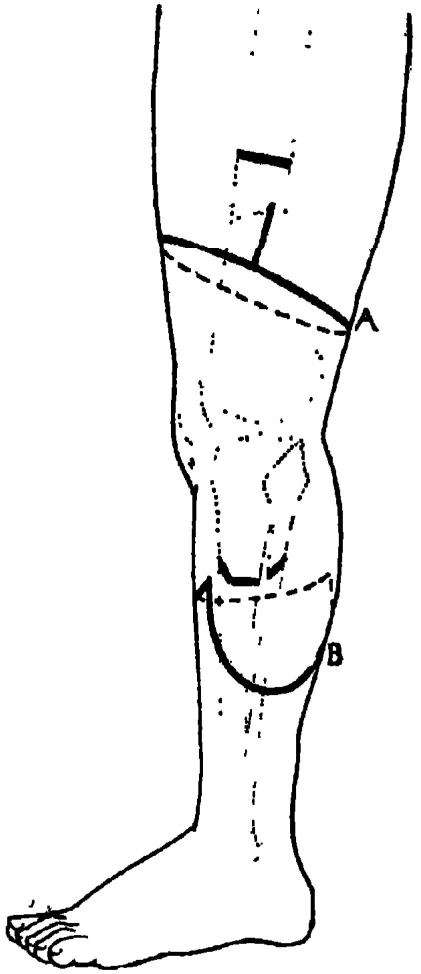


Fig. 192.—Amputations of the thigh and leg. A, Amputation through the lower part of the thigh by an elliptical incision; B, Amputation of the leg at the seat of election by a long external flap.

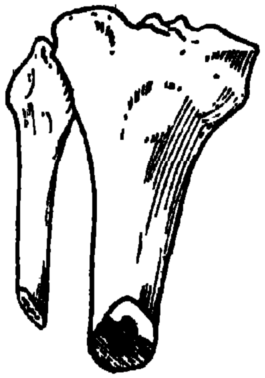


Fig. 193.—Showing how crest of tibia is bevelled off by an oblique cut.

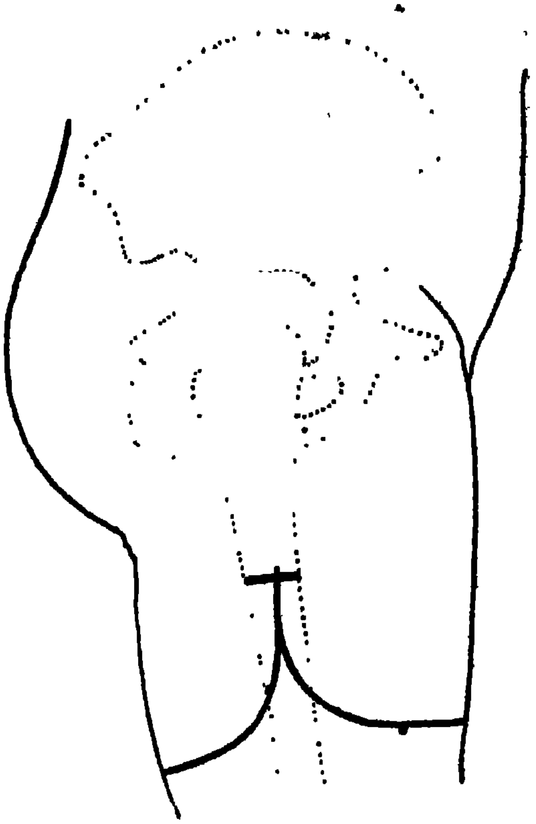


Fig. 194.—Amputation through the upper third of the thigh by equal flaps.

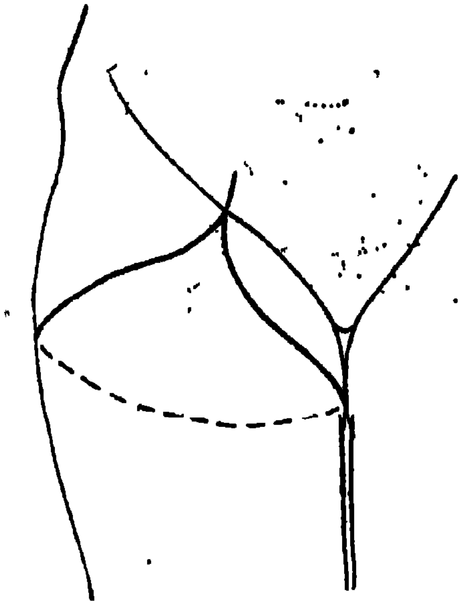


Fig. 195.—Amputation through the hip-joint by an anterior racket-shaped incision.

Special Amputations—Leg, ***continued.***

middle of the calf at an opposite point. The inner part of the leg is divided by a slightly curved incision joining the beginning and end of the flap incision (*Fig.* 192, B).

The bones are divided about 5 to 7 in. distal to the tubercle of the tibia. The fibula should be divided first, obliquely from above downwards and inwards. The tibia is divided obliquely from above downwards and backwards, so as to cut off the sharp angle of the crest (*Fig.* 193).

NEAR THE KNEE-JOINT.—Performed if there is not enough healthy tissue to give a stump 5 in. long. It is well worth while preserving the stump below the knee even if it is only 3 in. In this case it is better to remove the fibula entirely.

Through the Knee-joint.—This is not done now, because the best amputation through the knee will never give such a good stump for an artificial limb as one done through the lower third of the thigh. The limb-maker must have room to provide a hinge joint at the correct knee level.

Through the Thigh.—

LOWER OR MIDDLE THIRD.—The lower or middle third for the level of the bone division is the site of choice.

A circular or elliptical incision is made, a cuff of skin and fat is turned up, and the muscles are divided at a higher level.

The bone is divided at a level more than half the diameter of the thigh above the skin incision (*Fig.* 192, A).

UPPER THIRD.—In the upper third of a muscular thigh, the use of *equal flaps* is more convenient. Each flap is rather more than half the diameter of the limb, and includes skin, subcutaneous tissue, and fascia. The muscles are divided by a circular incision rather below the level of the bone section (*Fig.* 194).

Through the Hip-joint.—

Anterior Racket-shaped Incision—The handle of the racket is a vertical line midway between the anterior superior spine and the symphysis pubis. The circular part surrounds the thigh at the level of the ischial tuberosity (*Fig.* 195).

Hæmostasis.—The external iliac vessels are exposed, ligatured in two places, and divided. Pressure on the abdominal aorta at its point of bifurcation over the 4th lumbar vertebra controls bleeding from the other vessels.

Disarticulation.—The capsule of the joint is divided close to the acetabulum, the hip dislocated, and the muscles attached to the femur divided.

CHAPTER XLIX

SURFACE MARKINGS

HEAD AND NECK

BONY POINTS OF CRANIUM.—

NASION.—Nasofrontal suture.

GLABELLA.—Eminence above nasion.

INION.—External occipital protuberance.

BREGMA.—Point where coronal and sagittal sutures meet. The middle of vertical line joining the two pre-auricular points.

LAMBDA.—Junction of lambdoid and sagittal sutures, 2½ in. above and in front of inion.

PTERION.—Junction of frontal, parietal, sphenoid, and temporal bones; 1½ in. above centre of zygoma.

VENOUS SINUSES.—

SUPERIOR LONGITUDINAL.—From middle of glabella to inion; linear in front, ¾ in wide behind Inclines to the right.

LATERAL AND SIGMOID.—Band ½ in. wide from a point above inion to point ¾ in behind external auditory meatus, curving so that the highest point of curve is ¾ in above Reid's base-line.

MIDDLE MENINGEAL ARTERY.—

MAIN TRUNK AND ANTERIOR BRANCH.—From middle of zygoma to a point (pterion) 1½ in above middle of zygoma and 1½ in behind the external angular process of the frontal bone, then upwards and backwards at angle of 45°.

POSTERIOR BRANCH —From the main trunk backwards one finger's breadth above the zygoma.

CEREBRAL HEMISPHERE.—From glabella to pterion, curving downwards and backwards, ½ in. above orbital margin; from pterion to zygoma, curving downwards and forwards; along zygoma and line of lateral sinus to inion. Mesial border from inion to glabella.

FISSURE OF ROLANDO.—From point ½ in. behind middle of line from nasion to inion, downwards and forwards for 3⅜ in. at angle of 67½° (three-quarters of right angle); (or) downwards and forwards to a point 2 in. above pre-auricular point.

FACE CENTRES.—Lower third of fissure of Rolando.

ARM CENTRES.—Middle third of fissure of Rolando.

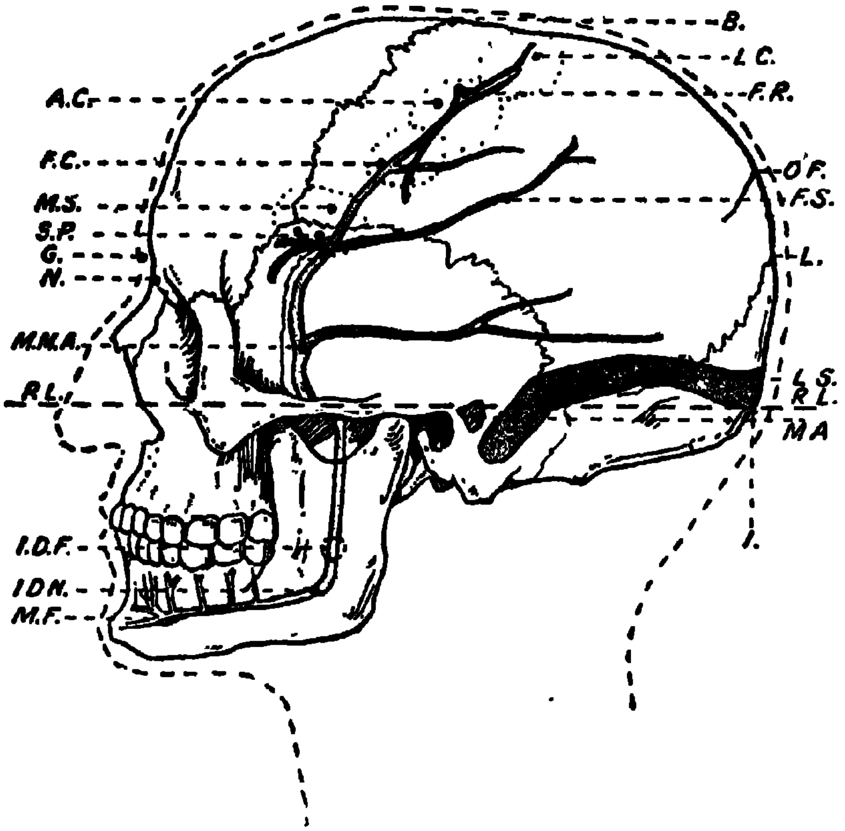
LEG CENTRES.—Upper third of fissure of Rolando.

MOTOR SPEECH CENTRE (Broca's convolution).—Just above the left Sylvian point.

THE SYLVIAN POINT.—1½ in. above the centre of the zygoma, and 1½ in. behind the external angular process It marks: (1) The pterion; (2) The middle meningeal artery; (3) The point of divergence of the anterior, ascending, and posterior limbs of the fissure of Sylvius; (4) The island of Reil; (5) The middle cerebral artery.

PLATE I

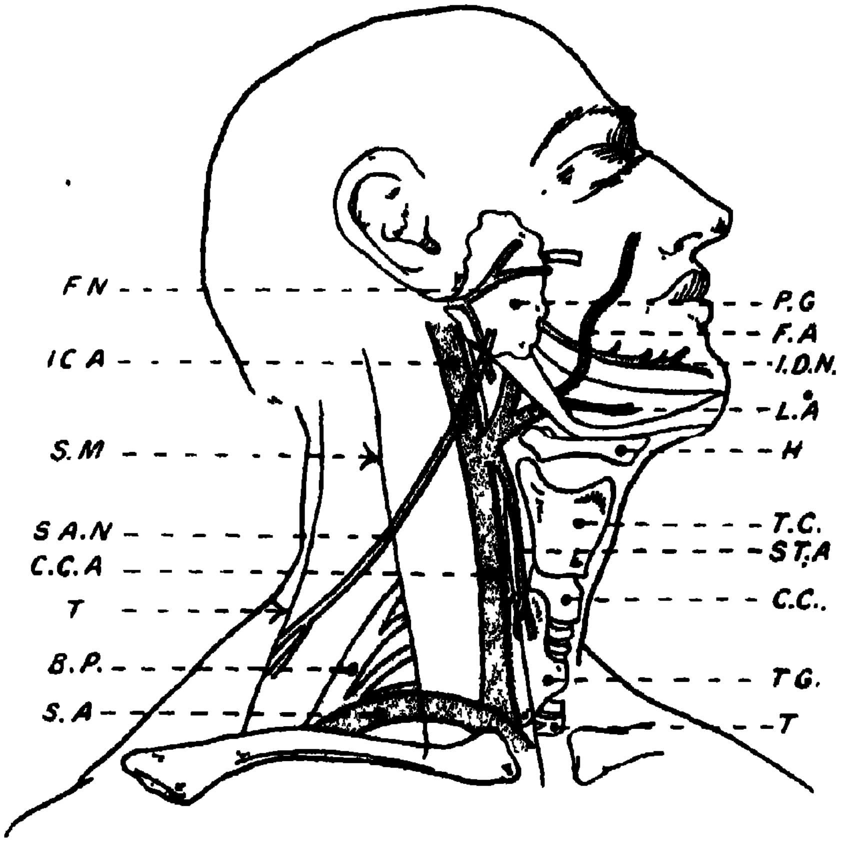
HEAD



B. Bregma; **A.C.** Arm centre; **F.C.** Face centre; **M.S.** Motor speech, **G.** Glabella; **N.** Nasion; **M.M.A.** Middle meningeal artery; **R.L.** Reid's line; **I.D.F.** Inferior dental foramen; **I.D.N.** Inferior dental nerve; **M.F.** Mental foramen; **L.C.** Leg centre; **F.R.** Fissure of Rolando; **O.F.** Occipito-parietal fissure; **L.** Lambda, **F.S.** Fissure of Sylvius; **S.P.** Sylvian point, pterion; **L.S.** Lateral sinus; **I.** Inion, **M.A.** Mastoid antrum.

PLATE II

FACE AND NECK



F.N. Facial nerve, I.C.A. Internal carotid, S.M. Sternomastoid; S.A.N. Spinal accessory nerve; C.C.A. Common carotid; T. Trapezius; B.P. Brachial plexus; P.G. Parotid gland; F.A. Facial artery; I.D.N. Inferior dental nerve, L.A. Lingual artery; H. Hyoid; T.C. Thyroid cartilage; S.T.A. Superior thyroid artery; C.C. Cricoid cartilage; T.G. Thyroid gland; S.A. Subclavian artery; T. Trachea.

Head and Neck, *continued.*

THE SYLVIAN FISSURE.—From the Sylvian point to a point ¾ in. below the parietal eminence.

THE OCCIPITO-PARIETAL FISSURE.—A line about 1½ in. long drawn outwards towards the external angular process of the frontal bone from a point three-quarters of the way between the glabella and inion.

REID'S BASE-LINE.—From lower margin of orbit to the external auditory meatus and on to inion.

TREPHINE POINTS.—

LATERAL SINUS.—¾ in. behind auditory meatus upon Reid's base-line.

MASTOID ANTRUM.—Suprameatal triangle, or point of junction of tangents drawn above and behind meatus.

TEMPOROSPHENOIDAL ABSCESS.—¾ in above post-auricular point.

CEREBELLAR ABSCESS.—1½ in. behind auditory meatus, 1 in below Reid's base-line.

LATERAL VENTRICLE.—1½ in. above external auditory meatus.

SUPRA-ORBITAL NOTCH, INFRA-ORBITAL FORAMEN, AND MENTAL FORAMEN.—Line joining junction of middle and inner thirds of supra-orbital ridge to interval between two lower bicuspid teeth, crosses each of these.

INFERIOR DENTAL NERVE.—May be exposed by a trephine midway between the anterior and posterior margins of the ramus on the level of the alveolar border.

FACIAL NERVE —Crosses ramus of the jaw level with the lower border of lobule of ear.

PAROTID DUCT —From lower margin of tragus or concha to point midway between ala nasi and angle of mouth. Ends on margin of masseter opposite second upper molar.

CAROTID ARTERY.—From sternoclavicular joint to midway between angle of jaw and tip of mastoid

COMMON CAROTID.—Up to the upper border of the thyroid cartilage.

EXTERNAL CAROTID —Same line above thyroid cartilage

SUPERIOR THYROID ARTERY, LINGUAL ARTERY, AND FACIAL ARTERY.—Run downwards, forwards, and upwards respectively from a point on the external carotid opposite the great cornu of the hyoid bone.

SUBCLAVIAN ARTERY.—Sternoclavicular joint to middle of clavicle. Forms a curve, which rises 1 in. above clavicle.

EXTERNAL JUGULAR VEIN.—From angle of jaw to middle of clavicle.

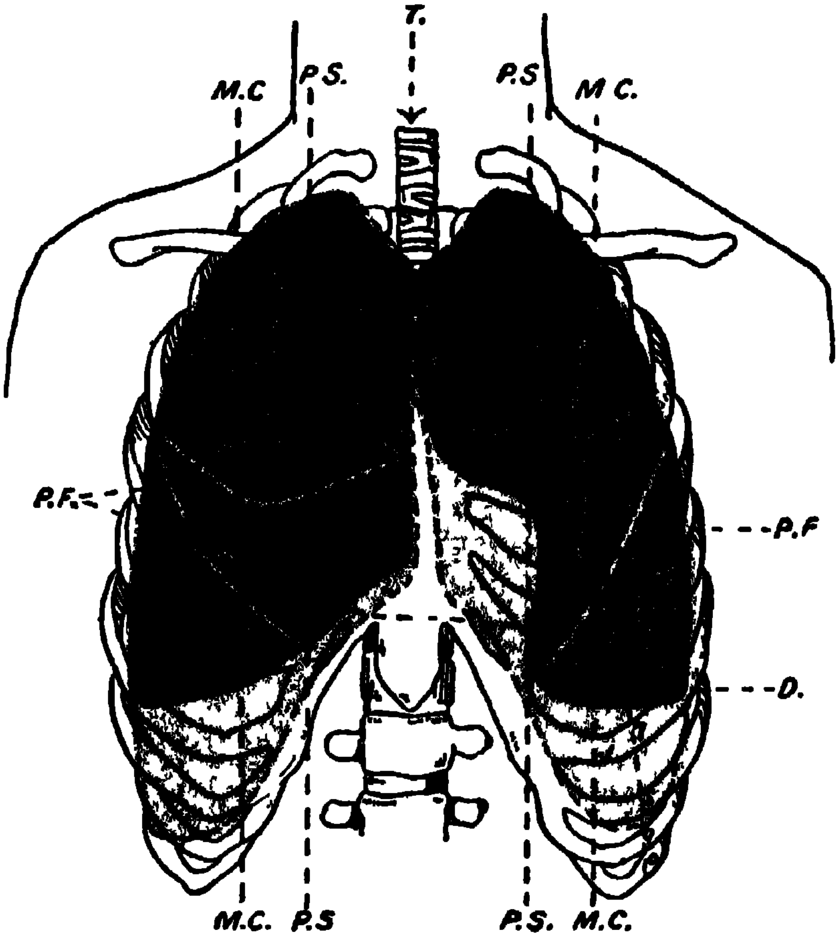
BRACHIAL PLEXUS.—Upper nerve from a point on the posterior border of the sternomastoid opposite the cricoid cartilage, to a point outside the middle of the clavicle. The lower nerve just above the clavicle.

SPINAL ACCESSORY NERVE.—From point between angle of jaw and mastoid to middle of posterior border of sternomastoid, then across posterior triangle to anterior border of trapezius.

UPPER BORDER OF THYROID CARTILAGE.—Level of disc between third and fourth cervical vertebræ.

PLATE III

LUNGS AND PLEURÆ



T. Trachea, P.S. Parasternal lines, M.C. Midclavicular lines, P.F. Pulmonary fissures, D. Diaphragm.

Purple = Lung *Blue* = Pleura.

Head and Neck, ***continued.***

CRICOID CARTILAGE.—Level of sixth cervical vertebra. Omohyoid crosses the carotid sheath. Beginning of œsophagus.

RIMA GLOTTIDIS.—Middle of the anterior border of the thyroid cartilage.

EPIGLOTTIS.—From below the thyroid notch to above the hyoid bone.

ISTHMUS OF THE THYROID GLAND.—A band ½ in. wide, which is ½ in. below the cricoid.

LATERAL LOBE OF THE THYROID.—Extends up to the thyroid cartilage, down to the clavicle, and outwards to the carotid line.

THORACIC VISCERA, Etc.

SURFACE LINES.—Parasternal line is a vertical line midway between the edge of the sternum and the midclavicular line.

LUNGS.—

Apex.—Extends 1 in. above inner third of clavicle.

Median Border.—

Right.—Sternoclavicular joint to middle of manubrium, to sixth costal cartilage in midline.

Left.—Sternoclavicular joint to middle of manubrium, to fourth costal cartilage; along this to parasternal line; down this to sixth costal cartilage.

Lower Border.—Sixth cartilage in parasternal line, to eighth rib in midaxillary line, to tenth rib in line of angle of scapula, to tenth dorsal spine (6–8–10).

Fissure.—Second dorsal spine downwards and forwards to sixth costal cartilage in parasternal line.

Transverse Fissure (on the right side only).—From main fissure forwards along fourth rib and cartilage.

Bifurcation of Trachea.—In front—the angle between the manubrium and body of the sternum (angulus Ludovici). Behind—between third and fourth dorsal spines.

Root of Lungs.—Opposite fourth, fifth, and sixth dorsal spines.

PLEURA.—

Apex.—1 in. above inner third of clavicle.

Median Border.—

Right.—From mid-manubrium to seventh costal cartilage in midline.

Left.—From mid-manubrium to sixth costal cartilage to left of midline.

Lower Border.—From lowest possible point of median border to 2 in. above tip of tenth costal cartilage, to twelfth rib, where it is crossed by erector spinæ, to twelfth dorsal spine; or more simply, seventh costal cartilage (parasternal) to ninth rib (mid-axilla) to eleventh rib (scapula angle) (7–9–11).

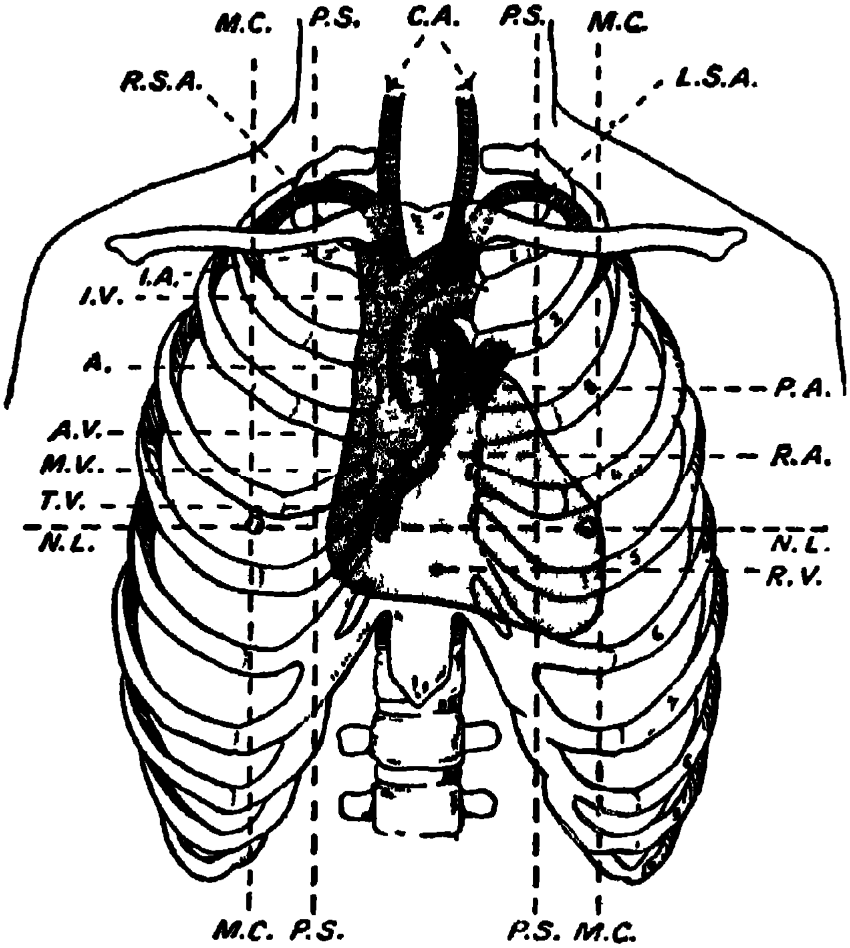
HEART AND PERICARDIUM.—Lie between curved lines joining four points:—

1. Second left intercostal space, upper border, 1 in. from sternum.
2. Second right intercostal space, lower border, ½ in. from sternum.
3. Sixth right costosternal junction, 1 in. from sternum.
4. Fifth left interspace, ½ in. inside mid-clavicular line.

Right Auriculoventricular Groove.—Joins 1 and 3.

PLATE IV

HEART AND GREAT VESSELS



C.A. Carotid arteries; **P.S.** Parasternal lines, **M.C.** Midclavicular lines, **R.S.A.** Right subclavian artery. **L.S.A.** Left subclavian artery. **I.A.** Innominate artery. **I.V.** Junction of right and left innominate veins to form superior vena cava, **A.** Aorta, **P.A.** Pulmonary artery; **A.V.** Aortic valve; **R.A.** Right auricle **M.V.** Mitral valve; **T.V.** Tricuspid valve, **N.L.** Nipple line; **R.V.** Right ventricle

Thorax, *continued.*

Valvular Orifices.—All below and to the left of the line 1–3.

In order from above downwards:—

Pulmonary and Aortic.—Opposite third costal cartilage.

Mitral.—Opposite fourth costal cartilage.

Tricuspid.—Opposite fourth and fifth costal cartilages.

Ascending Aorta.—From third left to second right chondrosternal joint.

Innominate Artery.—From middle of manubrium to right sternoclavicular joint.

First Portions of Left Subclavian and Left Carotid.—From middle of manubrium to left sternoclavicular joint.

Pulmonary Artery —From third to second left chondro-sternal junction.

Left Innominate Vein.—Left sternoclavicular joint to apex of the first right intercostal space.

Right Innominate Vein.—Right sternoclavicular joint to apex of the first right intercostal space.

Superior Vena Cava.—From first to the third right chondrosternal junction.

Internal Mammary Artery.—From sternoclavicular joint vertically downwards to the sixth costal cartilage, ½ in from sternum above, but closer to it below

Mammary Gland.—Extends up to the second rib, down to the sixth, inward to the border of the sternum, and outward to the mid-axillary line

The nipple lies on the fourth rib, or interspace, on the midclavicular line.

DIAPHRAGM—also highest level of liver (right), stomach (left).—

In Front (5, 5, 5) —Fifth left interspace in midclavicular line, to junction of ensiform cartilage with sternum (fifth sternal joint), to fifth right rib in midclavicular line.

Behind (8, 8, 8).—Eighth rib in the right scapular line, to eighth spine, to eighth interspace in the left scapular line.

ABDOMEN

SURFACE LINES.—

Transpyloric Plane, or Pyloric Line.—Horizontal plane midway between upper border of sternum and crest of pubes, and midway between ensiform cartilage and umbilicus. In normal parts it is also line joining tips of ninth costal cartilages.

It is the level of the following. Pylorus—first part of the duodenum. Gall-bladder. Duodenojejunal junction. Hila of kidneys—beginning of ureters. Body of pancreas. Beginning of mesentery. Lower border of first lumbar vertebra.

Subcostal Plane —Drawn through tips of the 10th ribs. Is on level with third lumbar vertebra.

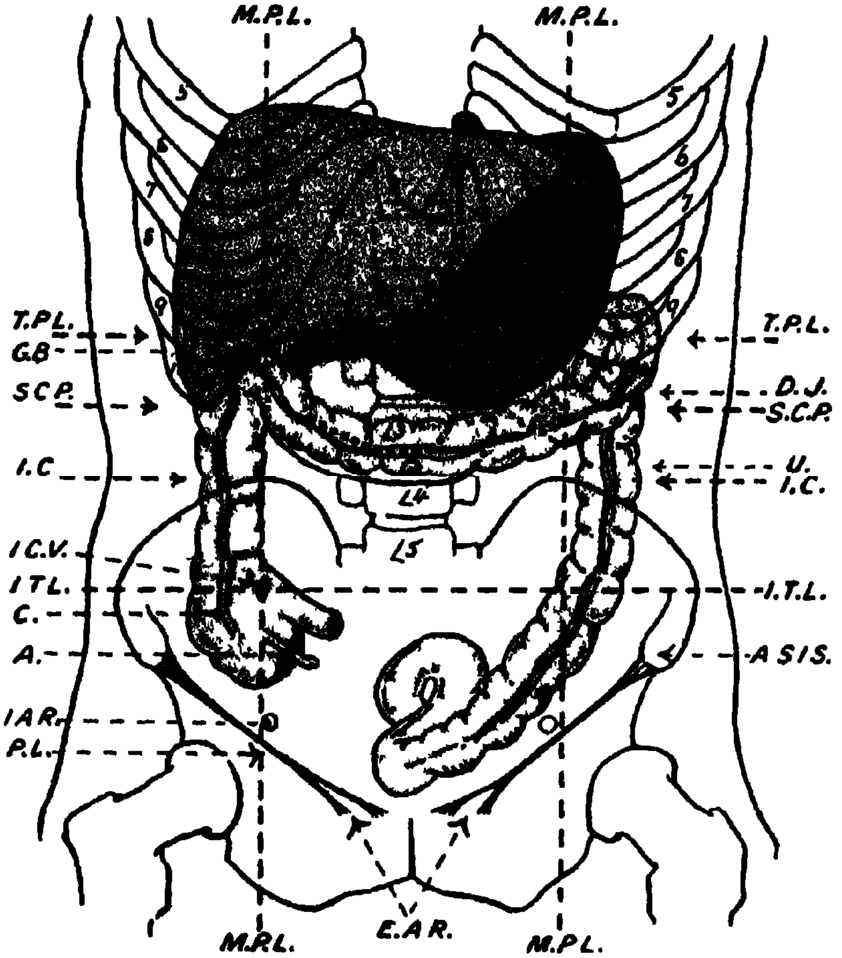
Line of Iliac Crests.—Level with fourth lumbar vertebra.

Intertubercular Plane.—Through the tubercles on outer lips of the iliac crests. Level with fifth lumbar vertebra.

Mid-Poupart Line.—Vertical line drawn through mid-point of Poupart's ligament.

PLATE V

ABDOMEN FROM FRONT



M.P.L. Mid-Poupart lines; **T.P.L.** Transpyloric line, **G.B.** Gall bladder; **D.J.** Points to duodenojejunal junction, **S.C.P.** Subcostal plane, **U.** Points to umbilicus, **I.C.** Line of the iliac crests, **I.C.V.** Ileocæcal valve; **I.T.L.** Intertubercular line, **C.** Cæcum; **A.** Appendix, **A.S.I.S.** Anterior superior iliac spine; **I.A.R.** Internal abdominal ring; **P.L.** Poupart's ligament, **E.A.R.** External abdominal ring.

Pink = Liver. *Blue* = Large intestine. *Purple* = Stomach.

Abdomen, ***continued.***

Linea Semilunaris.—From tip of the ninth costal cartilage to a point midway between the anterior iliac spine and the umbilicus, and thence to the pubic spine.

Lineæ Transversæ.—(1) At umbilicus; (2) Midway between umbilicus and xiphoid cartilage; (3) At xiphoid cartilage.

Internal Abdominal Ring.—Just above middle of Poupart's ligament.

External Abdominal Ring.—Just above the pubic crest.

LIVER.—

Above in Front.—From left fifth interspace in midclavicular line, to junction between sternum and ensiform cartilage, to fifth rib in the right midclavicular line.

Behind.—From eighth interspace in left scapular line, to eighth dorsal spine, to eighth rib in right scapular line.

Below.—From left fifth interspace in midclavicular line, to transpyloric plane in midline, to right costal margin.

STOMACH.—

Cardiac Orifice.—1 in. to left of junction of ensiform cartilage.

Pyloric Orifice.—1 in. to right of middle of transpyloric line.

Highest Level.—Fifth interspace in left mid-clavicular line.

Lowest Level.—Infracostal line.

DUODENUM.—Begins 1 in. to right of middle of transpyloric line. Ends 1 in. to left of middle of transpyloric line and a little below it. Extends to the right as far as right mid-Poupart line. Extends below as far as line joining highest points of the iliac crests.

MESENTERY.—From 1 in. to left of middle of transpyloric line and a little below it to a point midway along horizontal line between right anterior superior iliac spine and midline.

APPENDIX.—Attached end, midway along horizontal line between right anterior superior iliac spine and midline.

ILEOCÆCAL VALVE —Point where right mid-Poupart line crosses intertubercular line.

CÆCUM.—In right iliac fossa below the intertubercular plane, one-third inside and two-thirds outside the mid-Poupart line.

COLON.—

Both Sides Behind.—Vertical line ½ in. behind centre of iliac crest up to eleventh rib on the right side and to tenth rib on the left side.

Ascending Colon (in front).—From the intertubercular plane to the upper border of the ninth costal cartilage, lying outside but adjacent to the right mid-Poupart plane.

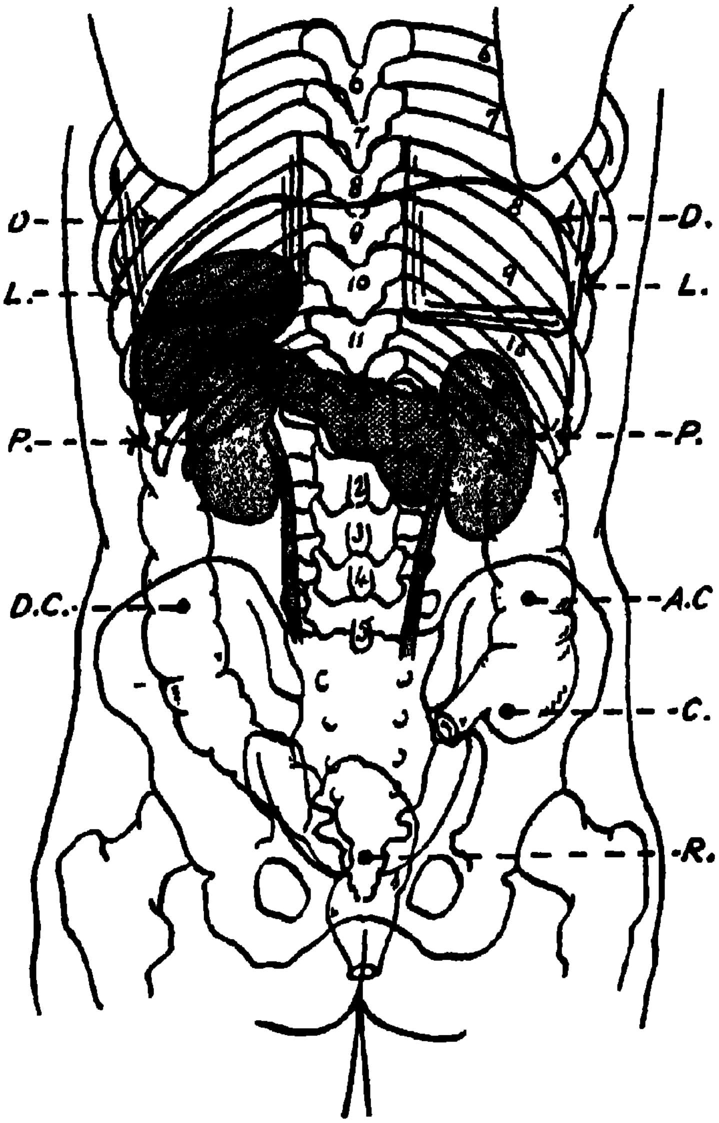
Transverse Colon (in front).—From the ninth right costal cartilage to the eighth left costal cartilage, where these are cut by the mid-Poupart plane. Curving downwards to the infracostal plane in the midline.

Descending Colon (in front).—From eighth left costal cartilage to the left iliac crest, outside but adjacent to the mid-Poupart plane.

RECTUM (behind).—From a point 1 in. below the level of the posterior superior iliac spine (which is on a level with the second sacral spine) to the anus.

PLATE VI

Abdomen from Behind



D Diaphragm **L** Lowest level of lungs, **P** Lowest level of pleuræ, **D C** Descending colon **A C** Ascending colon **C** Cæcum, **R** Rectum

Blue — Spleen *Pink* = Kidneys and ureters *Shaded* = Pancreas

Abdomen, ***continued.***

GALL-BLADDER.—At angle where ninth right costal cartilage is crossed by outer border of right rectus.

PANCREAS.—

Body lies on first lumbar vertebra, level of transpyloric line and of twelfth dorsal spine.

Tail from twelfth dorsal spine to the left, up to the tenth rib above pyloric line.

Head lies below pyloric line level with second lumbar vertebra.

SPLEEN.—Underlies ninth, tenth, and eleventh ribs on left side. Innermost point 1½ in. from tenth dorsal spine. Outermost point left mid-axillary line.

KIDNEYS.—

Behind.—From the level of eleventh dorsal spine to that of third lumbar spine. Hilum 2 in. from first lumbar spine. Inner border 2 in. from midline, outer border 4 in. from midline.

In Front.—Hilum is on transpyloric line 2 in. from middle line (tip of the ninth costal cartilage). Thence the kidney extends upwards 2 in. (to the sixth costal cartilage on the left and to the seventh on the right) Outwards 2 in. Downwards 2 in. to the infracostal plane on the left, and below this on the right. In each case the upper pole is ½ in. nearer the midline than the lower Left kidney is ½ in. higher than right

Ureter.—From hilum to posterior superior iliac spine behind. From hilum to bifurcation of iliac arteries in front.

Suprarenal Capsule.—Opposite inner part of eleventh intercostal space.

THE BLOOD-VESSELS.—

Abdominal Aorta.—A band 1 in. wide from the tip of the xiphoid cartilage to the line joining the highest points of the iliac crests. Lying to the left of the midline

Inferior Vena Cava.—A band 1 in. wide below and 1½ in wide above, from the line joining the highest points of the iliac crests to the apex of the fifth right intercostal space. Lying to the right of the midline Perforates the diaphragm at its highest point opposite the eighth dorsal spine.

The Cœliac Axis and Superior Mesenteric Arteries arise just above the transpyloric plane.

The Renal Arteries arise just below the transpyloric plane

The Inferior Mesenteric Artery arises just above the infracostal plane

Iliac Artery.—From the left of the middle of line joining highest iliac points to a point midway between the anterior superior iliac spine and the symphysis pubis.

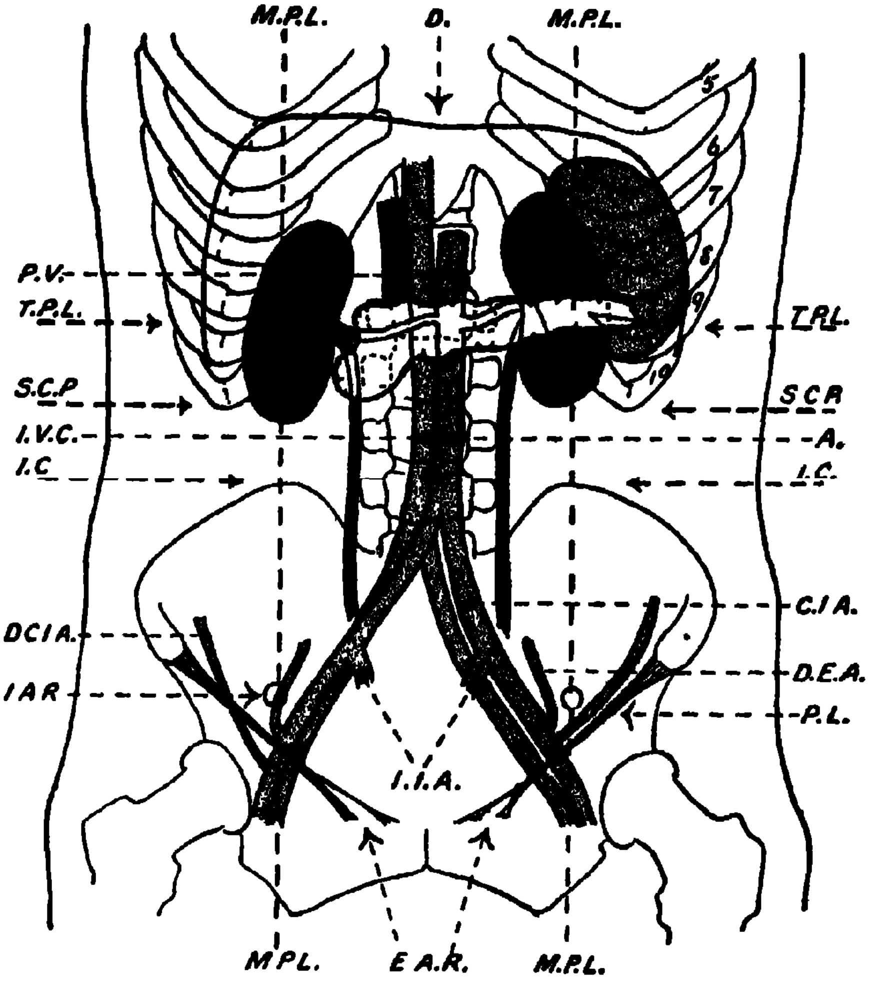
Upper third = common iliac, lower two-thirds = external iliac.

Deep Epigastric Artery.—From a point midway between the anterior superior iliac spine and the symphysis pubis, towards the umbilicus.

Deep Circumflex Iliac Artery.—Just above the outer half of Poupart's ligament and round the iliac crest.

PLATE VII

Deep Abdominal Viscera and Vessels from Front



M P L Mid Poupart line, **D** Diaphragm, **P V** Portal vein, **T P L** Transpyloric line, **S C P** Subcostal plane **I V C** Inferior vena cava, **I C** Line of the iliac crests, **A** Aorta, **C I A** Common iliac artery, **D C I A** Deep circumflex iliac artery, **D E A** Deep epigastric artery, **I A R** Internal abdominal ring, **P L** Poupart's ligament, **I I A** Internal iliac artery, **E A R** External abdominal ring

Abdomen, ***continued.***

THE PORTAL VEIN THE COMMON BILE-DUCT THE HEPATIC ARTERY THE FORAMEN OF WINSLOW	From a point 1 in. to the right of the middle of the transpyloric line upwards and a little to the right to the costal margin. The vein and bile-duct extend 2 in. below the point given; the vein to the left and the duct to the right.

THE SPINAL CORD.—Extends down as far as the first lumbar spine behind, or the transpyloric line in front. The spinal dura mater extends down to ¼ in. below the posterior superior iliac spines.

UPPER EXTREMITY

ACROMIOCLAVICULAR JOINT.—Runs in a sagittal direction midway between the outer and inner borders of the arm when it is hanging at the side. It is just outside a bony tubercle on the anterior margin of the clavicle.

CORACOID PROCESS.—On the anterior deltoid margin, 1 in. below the junction of the middle and outer thirds of the clavicle.

BICIPITAL GROOVE.—A line 2 in. long downwards from the tip of the acromion in the long axis of the humerus

TUBEROSITIES.—With the arm everted, the lesser tuberosity lies between the coracoid process and the bicipital groove. The great tuberosity lies outside the bicipital groove.

HEAD OF THE HUMERUS looks in the same direction as the internal epicondyle

HUMERAL EPICONDYLES lie on same level as tip of the olecranon when the arm is extended

RADIO-HUMERAL JOINT is ¾ in. below the tip of the external epicondyle.

ULNAR-HUMERAL JOINT is 1 in. below the tip of the internal epicondyle.

THE EXTERNAL INTERMUSCULAR SEPTUM extends from the insertion of the deltoid to the external epicondyle

THE INTERNAL INTERMUSCULAR SEPTUM extends from the insertion of the coracobrachialis to the internal epicondyle.

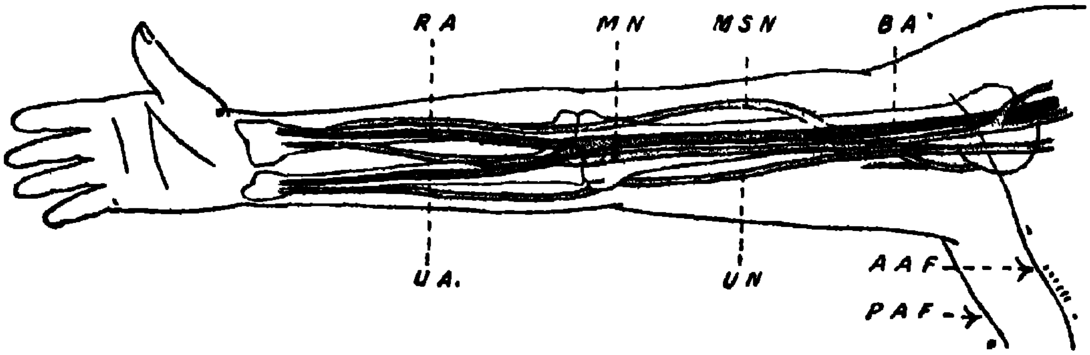
AXILLARY AND BRACHIAL ARTERIES.—With the arm abducted and the hand supinated, the line from the centre of the clavicle to the point midway between the humeral condyles and 1 in. below them, represents the axillary and brachial arteries. A point one-third of the distance from the outer end of the anterior to the outer end of the posterior axillary folds is the end of the axillary and the beginning of the brachial.

THE RADIAL ARTERY.—From a point midway between the two epicondyles and 1 in. below them to the radial side of the tendon of the flexor carpi radialis. Thence under the tendons of the extensor ossis metacarpi pollicis and extensor brevis pollicis to the back of the first metacarpal space at its apex.

THE ULNAR ARTERY.—From a point midway between the epicondyles and 1 in. below them to the junction of the upper and middle thirds of a line drawn from the internal epicondyle to the radial side of the pisiform bone, and thence down this line.

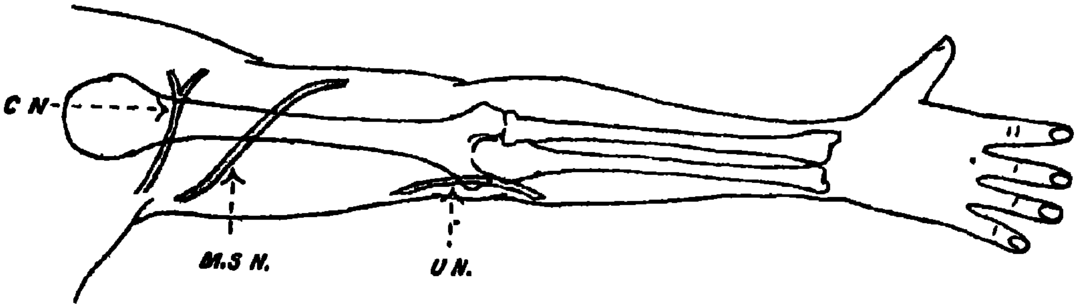
PLATE VIII

ARM FROM FRONT



R A Radial artery, **M N** Nedian nerve **M S N** Musculospiral nerve, **B A** Brachial artery, **U A.** Ulnar artery, **N** Ulnar nerve, **A A F** Anterior axillary fold, **P A F** Posterior fold.

ARM FROM BEHIND



C N Circumflex nerve **M S N** Musculospiral nerve **U N** Ulnar nerve

Upper Extremity, ***continued.***

THE SUPERFICIAL PALMAR ARCH is convex downwards and reaches to a level with the web of the thumb.

THE DEEP PALMAR ARCH is one finger's breadth above the superficial,

THE MEDIAN BASILIC AND MEDIAN CEPHALIC VEINS run from the centre of the antecubital space to the inner and outer borders of the belly of the biceps respectively.

THE BASILIC VEIN runs up the inner border of the biceps, and pierces the deep fascia rather more than halfway up the arm.

THE CEPHALIC VEIN runs up the outer border of the biceps, and then between the deltoid and pectoral muscles, and pierces the costocoracoid membrane.

THE SUPERFICIAL LYMPHATICS.—Run with the superficial veins. and end in trunks which accompany the basilic and cephalic veins.

THE LYMPHATIC GLANDS.—

1. AT THE BEND OF THE ELBOW—supratrochlear and antecubital glands draining the front of forearm and its inner side.
2. AXILLARY SET—lying with the axillary vein on the outer side of the axilla and draining forearm.
3. PECTORAL SET—along the outer margin of the pectoralis minor, and draining the chest wall and breast
4. SUBSCAPULAR GROUP—along the posterior fold of the axilla, draining the back, the axilla, and part of the mammary gland
5. SUBCLAVIAN GROUP—lying beneath both pectoral muscles below the clavicle, and running up under it to the posterior triangle of the neck. They receive the efferents from all the other groups.

THE CIRCUMFLEX NERVE (and posterior circumflex artery) —Passes round the back of the humerus just above the centre of the deltoid muscle.

MUSCULOSPIRAL NERVE (and superior profunda artery).—From the junction of the arm and posterior axillary fold to a point one-third of the way from the deltoid insertion to the external epicondyle, and thence in the groove between the biceps and supinator longus.

RADIAL NERVE.—From the groove between the biceps tendon and the supinator longus to the junction of the middle and lower thirds of the arm, when it passes beneath the supinator to the back of the wrist.

MEDIAN NERVE —In the arm the same as the brachial artery; at the elbow it lies on its inner side, in the forearm it runs down to the wrist, when it passes beneath the anterior annular ligament to the ulnar side of the flexor carpi radialis.

ULNAR NERVE —In the arm, at first with the brachial artery, then from the junction of the upper and middle thirds it runs to the groove behind the internal condyle, and thence to the radial side of the pisiform bone.

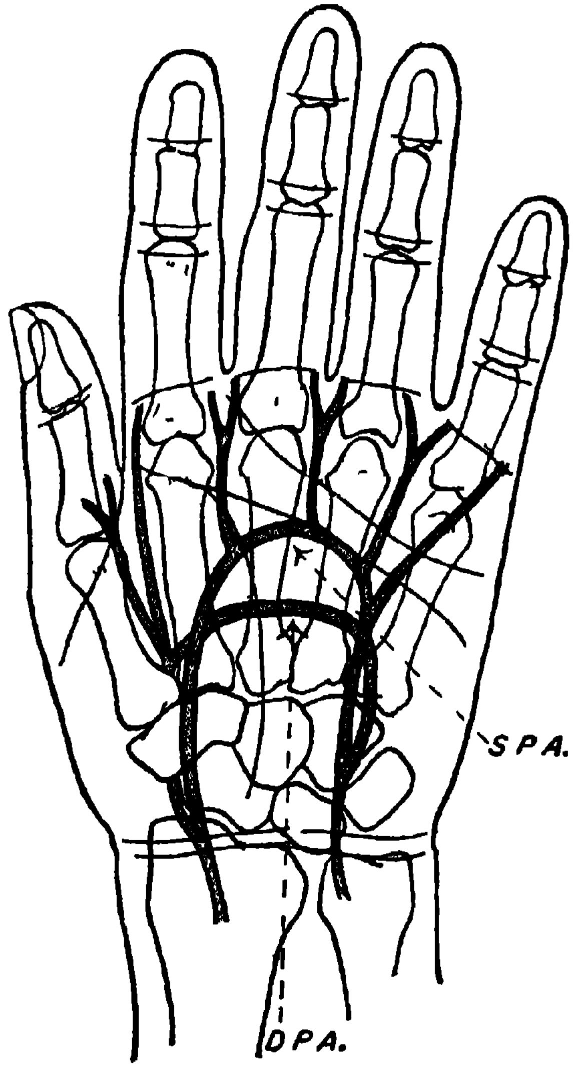
THE ELBOW-JOINT.—One finger's breadth below the anterior skin crease.

THE WRIST-JOINT.—Indicated by the upper of the two skin creases in front.

THE TIP OF THE RADIAL STYLOID PROCESS.—In the 'anatomical snuffbox', between the tendons of the flexores primi and secundi internodii pollicis It is on a level with the lower wrist skin crease, ½ in. below the wrist-joint.

PLATE IX

HAND FROM FRONT



D P A Deep palmar arch, S P A Superficial palmar arch

Upper Extremity, *continued.*

THE TIP OF THE ULNAR STYLOID is just below the level of the upper wrist skin crease, i.e., it is at ¼ in. higher level than the radial styloid.

THE TUBERCLE OF THE SCAPHOID is between the tendons of the flexor carpi radialis and the extensor ossis metacarpi pollicis on the lower wrist crease.

THE ANTERIOR ANNULAR LIGAMENT is 1 in. wide; its upper border corresponds to the lower wrist skin crease, and it stretches from the pisiform and unciform bones to the scaphoid and trapezium.

THE COMMON FLEXOR SYNOVIAL SHEATH (which includes the sublimis and profundus tendons and median nerve), extends two or three finger-breadths above the wrist-joint and below to the upper transverse palmar crease. It extends also into the little finger. The flexor longus pollicis has a separate sheath.

THE FLEXOR SHEATHS OF THE THREE MIDDLE FINGERS extend up as far as the lower palmar skin crease.

THE METACARPOPHALANGEAL JOINTS lie halfway between the lower palmar skin crease and the web of the fingers.

THE INTERPHALANGEAL JOINTS lie between the skin creases of the middle joint and opposite the crease of the lower.

THE TUBERCLE AT THE BACK OF THE RADIUS lies on the outer side of the sheath of the extensor secundi internodii pollicis.

THE TUBERCLE AT THE BACK OF THE CARPUS is the styloid process of the second metacarpal.

THE POSTERIOR ANNULAR LIGAMENT is 1 in. broad, and extends from the back of the radius to the interval between the ulna and the carpus.

THE SYNOVIAL SHEATHS AT THE BACK OF THE WRIST extend about 1 in. above and below the annular ligament They are:—

1. For the extensores ossis metacarpi and primi internodii pollicis.
2. For the extensores longus and brevis carpi radialis on the floor of the 'anatomical snuffbox.'
3. For the extensor secundi internodii pollicis on the ulnar side of the radial tubercle.
4. For the extensores indicis and communis digitorum behind the radius.
5. For the extensor minimi digiti between the radius and ulna.
6. For the extensor carpi ulnaris between the head and styloid process of the ulna.

LOWER EXTREMITY

HIGHEST POINT OF ILIAC CREST.—On a level with the spine and body of fourth lumbar vertebra.

TUBERCULAR POINT.—2½ in. behind the anterior superior spine. Level with fifth lumbar vertebra

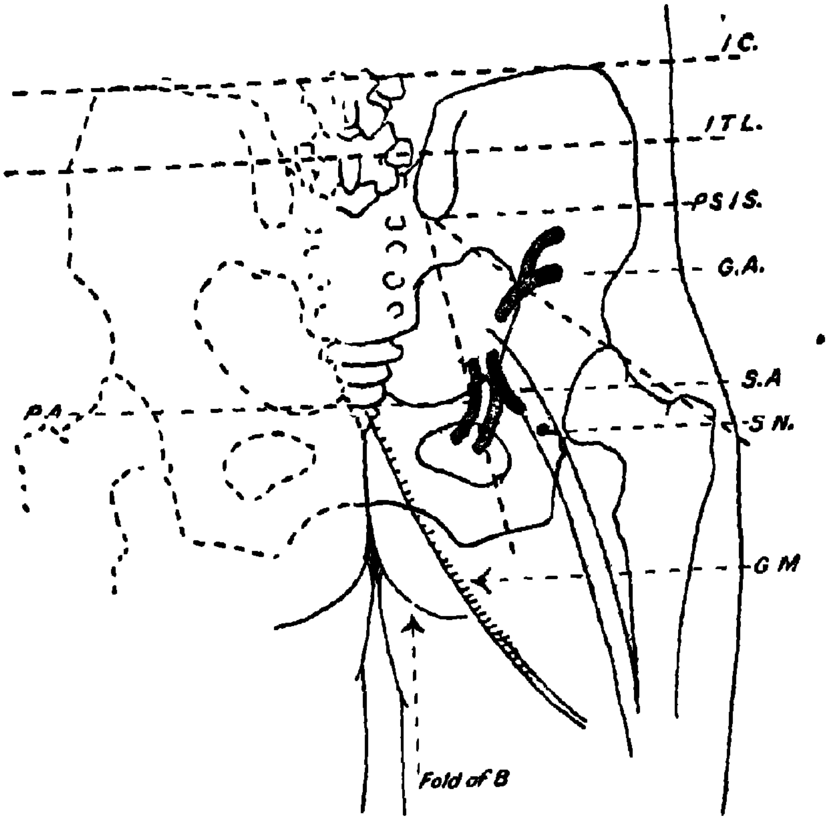
POSTERIOR SUPERIOR ILIAC SPINE.—Level with second sacral spine. Middle of sacro-iliac joint.

GREAT TROCHANTER.—Tip is crossed by Nélaton's line from anterior superior iliac spine to the ischial tuberosity. It is vertically below the tubercular point.

BRYANT'S TRIANGLE.—Patient lying horizontal. Draw a horizontal line up from tip of trochanter. Drop a perpendicular line from the anterior superior spine to meet the first line. Join the anterior superior

PLATE X

GLUTEAL REGION



I C Highest point of iliac crests, **I T L.** Level of iliac tubercles, **P S I S** Posterior superior iliac spine, **G A** Gluteal artery, **S A** Sciatic artery, **P A** Pubic artery, **S N.** Sciatic nerve, **G M** Gluteus maximus, **Fold of B.** Fold of buttock. The two broken lines leading downward and outward from the posterior superior iliac spine indicate the surface markings for the gluteal and for the sciatic and pudic arteries

Lower Extremity, *continued.*

spine to the tip of trochanter. Horizontal line shows the length of the femoral neck. Vertical line shows degree of rotation.

In the erect position tip of trochanter is vertically below tubercle on iliac crest.

SMALL TROCHANTER.—Is felt above the outer end of the gluteal fold when the femur is internally rotated.

LOWER BORDER OF THE GLUTEUS MAXIMUS.—Join junction of upper and middle thirds of the femur to the middle of the gluteal fold and produce upwards.

GLUTEAL ARTERY —Leaves the pelvis at a point at the junction of the upper and middle thirds of a line joining the posterior superior spine to the tip of the great trochanter.

SCIATICA AND PUDIC VESSELS.—Leave the pelvis at a point at the junction of the middle and lower thirds of a line joining the posterior superior spine to the ischial tuberosity.

GREAT SCIATIC NERVE.—Vertical line midway between the great trochanter and the ischial tuberosity.

EXTERNAL INTERMUSCULAR SEPTUM OF THE THIGH.—Line joining the iliac tubercle to the head of the fibula.

TENDON OF THE BICEPS —External hamstring.

TENDON OF THE SEMITENDINOSUS.—Internal hamstring.

TENDON OF THE SEMIMEMBRANOSUS.—Lies deep to the internal hamstring and then winds round underneath the inner head of the gastrocnemius; a bursa lying between the two tendons.

HIP-JOINT is marked by a circle inscribed in the following triangle: Vertical line dropped from the anterior superior spine, horizontal line drawn out from the pubic spine, Poupart's ligament.

PUBIC SPINE is felt:—

In the male, by invagination of the scrotum into the external ring

In the female, by following up the tendon of the adductor longus.

SAPHENOUS OPENING.—1½ in. below and to the outer side of the pubic spine.

FEMORAL ARTERY.—Flex the hip and knee and rotate the thigh outwards. Join the mid-point between the anterior superior iliac spine and the symphysis pubis to the most prominent part of the internal condyle. Upper two inches represent the common femoral, the rest the superficial.

Upper third of line represents the artery in Scarpa's triangle. Middle third of line represents the artery in Hunter's canal. Lower third of line represents the popliteal artery in popliteal space.

POPLITEAL ARTERY BEHIND.—From apex of the popliteal space to midway between the two condyles at a point on a level with the tibial tubercle.

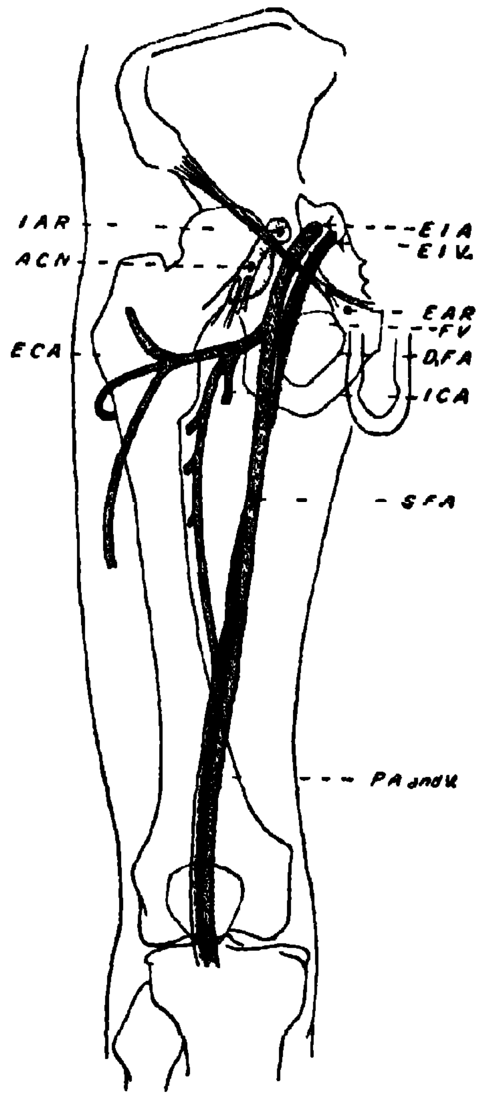
ANTERIOR TIBIAL ARTERY —From a point midway between the head of the fibula and the external tibial tuberosity to a point midway between the malleoli, where it is crossed by the extensor longus hallucis.

DORSALIS PEDIS.—From midway between the malleoli to the base of the first metatarsal space. It is crossed by the inner tendon of the extensor brevis digitorum.

POSTERIOR TIBIAL.—From a point midway between the femoral condyles, but on a level with the tibial tubercle, down to a point one finger-breadth behind the inner malleolus.

PLATE XI

VESSELS AND NERVES OF THIGH



E I A External iliac artery **I A R** Internal abdominal ring **E I V** External iliac vein, **A C N** Anterior crural nerve **E A R** External abdominal ring **F V** Femoral vein **D F A** Deep femoral artery **E C A** External circumflex femoris artery **I C A** Internal circumflex femoris artery **S F A** Superficial femoral artery **P A V** Popliteal artery and vein

Lower Extremity, *continued.*

INTERNAL PLANTAR ARTERY.—From the tip of the inner malleolus to the ball of the great toe.

EXTERNAL PLANTAR ARTERY.—From the tip of the inner malleolus to the base of the fifth metatarsal bone, and thence to the base of the first metatarsal cleft.

INTERNAL SAPHENOUS VEIN AND NERVE.—From in front of the inner ankle to the groove between the sartorius insertion and the inner head of the gastrocnemius, behind the inner condyle of the femur, and up to the saphenous opening.

EXTERNAL SAPHENOUS VEIN AND NERVE.—From behind the outer ankle to the middle of the popliteal space.

THE SUPERFICIAL LYMPHATICS follow the same line as the above two veins.

THE LYMPHATIC GLANDS.—

1. Popliteal Group—generally one or two, drain the outer side of the leg or foot.
2. Inguinal Group—six to seven, parallel to Poupart's ligament, drain the anterior abdominal wall and upper part of the thigh.
3. Pubic Set —two to three, over the pubis, drain the buttocks, anus, perineum, and external genitals.
4. Superficial Femoral—two to three, on the inner side of the femoral vessels at the saphenous opening, drain the greater part of the thigh and inner side of the leg and foot.
5. Deep Femoral—one or two lie in the crural canal, and drain all the other superficial groups, together with the deep parts of the leg.

ANTERIOR CRURAL NERVE.—Downwards from a point midway between the anterior superior iliac spine and the spine of the pubes.

FEMORAL RING AND CRURAL CANAL.—Lie on the outer side of the pubic spine and on the inner side of the femoral vessels.

ADDUCTOR TUBERCLE.—Just above the most prominent part of the internal condyle. In the interval between the vastus internus in front and the sartorius behind. It marks the site of the epiphysial junction.

KNEE-JOINT.—In full extension the lower border of the patella is on a level with the joint. In semiflexion a triangle exists in front of the inner side, bounded by the inner border of the patella, anterior lower border of the internal femoral condyle, and upper border of the internal tibial tuberosity. In this a displaced internal semilunar cartilage, or loose body, can be felt.

THE HEAD OF THE FIBULA is on the same level as the tubercle of the tibia.

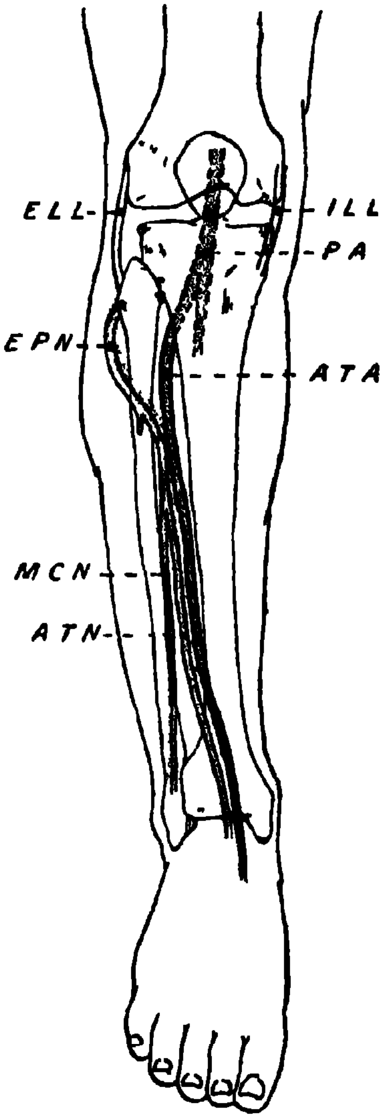
INTERNAL LATERAL LIGAMENT OF THE KNEE.—From the most prominent point of the internal condyle to the inner surface of the inner tibial tuberosity.

EXTERNAL LATERAL LIGAMENT.—From the most prominent point of the external condyle to the tip of the head of the fibula.

EXTERNAL POPLITEAL NERVE.—Behind the tendon of the biceps. Winds forwards round the neck of the fibula.

PLATE XII

VESSELS AND NERVES OF LEG



E L L External lateral ligament, **I L L** Internal lateral ligament, **P.A.** Popliteal artery, **E P N** External popliteal nerve, **A T A** Anterior tibial artery **M C N** Musculo-cutaneous nerve, **A T N** Anterior tibial nerve

Lower Extremity, ***continued.***

POSTERIOR PERONEAL INTERMUSCULAR SEPTUM.—Line from the posterior border of the head of the fibula to the posterior border of the external malleolus. Indicates the line to cut down on the fibula.

ANTERIOR PERONEAL INTERMUSCULAR SEPTUM.—Line from the anterior border of the head of the fibula to the anterior border of the external malleolus.

MUSCULOCUTANEOUS NERVE (cutaneous part).—Lower half of the above line.

ANKLE-JOINT AND MALLEOLI.—The joint lies ½ in. above the tip of the internal malleolus and 1 in. above that of the external; the external malleolus being ½ in. below and behind internal.

PERONEAL TUBERCLE.—½ in. below the tip of the external malleolus; the tendon of the peroneus brevis lies above this, and that of the peroneus longus below. Each has a synovial sheath, which join above the tubercle, and thence run 1 in. above the outer ankle.

EXTENSOR BREVIS DIGITORUM lies in the interval between the tendons of the peroneus brevis and tertius.

HEADS OF THE ASTRAGALUS AND OS CALCIS are felt in the same interval

THE HEAD OF THE ASTRAGALUS is also felt in the interval between the tibialis anticus and posticus tendons, and it ought to lie above the line joining the tip of the internal malleolus to the tubercle of the scaphoid.

SUSTENTACULUM TALI is ½ in. below the tip of the internal malleolus. Between the two bony points run the tendons of the tibialis posticus and the flexor longus digitorum; below the sustentaculum runs the flexor longus hallucis

TARSO-METATARSAL JOINT OF THE GREAT TOE is halfway between the metatarso-phalangeal joint and the tip of the inner malleolus

THE TUBERCLE OF THE SCAPHOID is halfway between the tarso-metatarsal joint of the great toe and the inner malleolus.

THE ASTRAGALO-CALCANEAN JOINT on the inner side is just above the sustentaculum tali, and on the outer side midway between the tip of the external malleolus and the peroneal tubercle.

THE CALCANEO-CUBOID JOINT is midway between the peroneal tubercle and the base of the fifth metatarsal bone.

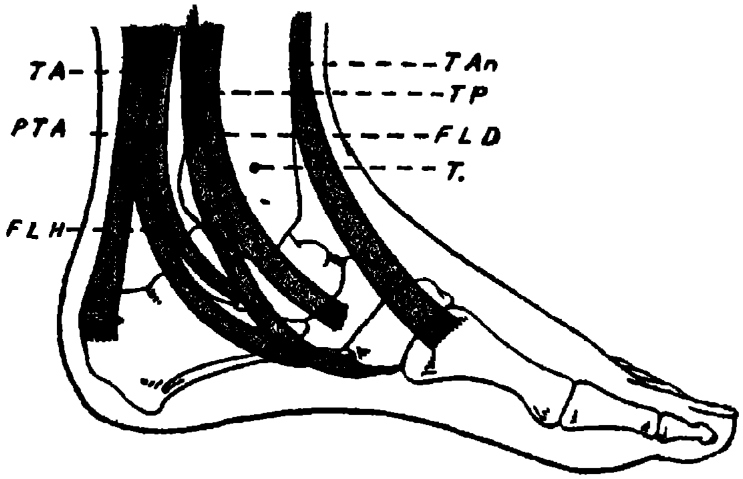
ANTERIOR ANNULAR LIGAMENT:—

1. UPPER PART.—A band two finger-breadths wide from the anterior margin of the tibia to that of the fibula at their lower ends. Has one synovial sheath, that for the tibialis anticus.
2. LOWER PART —A Y-shaped band from the os calcis in front of the tip of the external malleolus up to the anterior border of the tibia and across to the inner margin of the instep. Has three synovial sheaths, for the tibialis anticus, extensor longus hallucis, and extensor communis with peroneus tertius.

INTERNAL ANNULAR LIGAMENT.—From the tip of the inner malleolus to the inner side of the posterior process of the os calcis. Has three synovial sheaths beneath it from within outwards for: (1) The tibialis posticus; (2) The flexor longus digitorum; (3) The flexor longus hallucis. All of these extend about 1 in. above the malleolus.

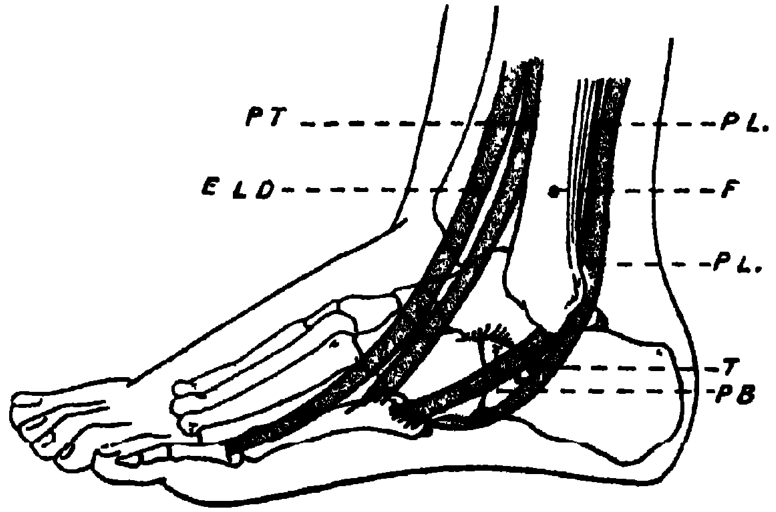
PLATE XIII

FOOT FROM INNER ASPECT



T An Tibialis anticus, **T A** Tendo Achillis, **T P** Tibialis posticus **P T A** Posterior tibial artery **F L D** Flexor longus digitorum, **T** Tibia, **F L H** Flexor longus hallucis

FOOT FROM OUTER ASPECT



P T Peroneus tertius, **P L** Peroneus longus, **E L D** Extensor longus digitorum, **F** Fibula, **T** Peroneal tubercle, **P B** Tendon of peroneus brevis

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The letters s.m. are an abbreviation for 'Surface Markings.' Surface Markings are illustrated in *Plates I–XIII* at the end of the book; references to these plates have only been made in the more important entries in the Index.

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